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INHERENT HEALING PROPERTIES OF ABSCESS OF THE BRAIN

CLINICO-ANATOMIC SURVEY OF FIFTEEN VERIFIED CASES

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The diagnosis and the precise localization of an abscess of the brain offer few difficulties in the typical case. The difficulties are, of course, more numerous in the atypical instance, but even there the diagnostic obstacles are not insurmountable. However, even when an abscess is diagnosed and localized, there arises the problem of devising a satisfactory method of therapeutic approach. The old axiom to evacuate pus wherever found, of course, holds also for an abscess of the brain, but attempts to carry out this measure are not frequently rewarded with success. The number of cured abscesses of the brain is still too small except for the isolated instances of satisfactory results in the hands of a few surgeons. Is this not due to the lack of a uniform and scientifically well grounded surgical procedure? It is true that several attempts have been made to depart from the time-worn, inadequate method of blindly searching for the abscess and the more or less crude means of evacuating and draining it. We have King's [1] method and, more recently, the method of Dandy,[2] but neither procedure has been sufficiently tested to warrant acceptance as the standard means of approach in the surgical treatment of abscess of the brain. The question arises whether a uniform surgical approach has not been evolved

Read in part before the Section of Otology of the New York Academy of Medicine, Nov. 12, 1926.

From the Neurological and the Otological Services and Division of Laboratories, Mount Sinai Hospital.

1. King, Joseph E. J.: The Treatment of Brain Abscess by Unroofing and Temporary Herniation of Abscess Cavity with the Avoidance of the Usual Drainage Method, Surg., Gynec. & Obst. **39**:554, 1924.

2. Dandy, Walter E.: Treatment of Chronic Abscess of the Brain by Tapping: Preliminary Note, J. A. M. A. **87**:1477 (Oct. 30) 1926.

for lack of precise knowledge of the life cycle of abscess of the brain. Suppurative processes elsewhere in the body are generally treated more successfully, very likely because of a better knowledge of the morphologic alterations occurring in the course of the evolution of a circumscribed suppurative lesion. The evacuation of a poorly localized and not fully encapsulated suppurative focus is not looked on with favor, for there the results of premature surgical intervention are only too well known. Are precautions against spread of the inflammatory process by poorly timed and by inadequate surgical management less essential in the suppurative processes of the brain? Or, are they not more imperative because of the paucity of connective tissue in the brain and the slowness with which suppurative processes in the brain encapsulate? It is quite obvious that the rational treatment of a circumscribed suppurative focus in the brain, as elsewhere, depends on the precise knowledge of the most opportune time for successful evacuation of the abscess, accurate knowledge of the precautions that are to be taken against spread of the infection and the measures best adapted for more complete evacuation of pus and stimulation of a healing process. The acquisition of such knowledge depends on a better understanding of the character of the lesion, of the various stages in its evolution and of its inherent potencies for self-limitation. It is with this in mind that we present our material, which consists of fifteen cases of abscess of the brain, varying in location and in the degree of chronicity. It is our aim to add a few observations on the morphologic features of abscess of the brain, to correlate them with carefully noted clinical manifestations and, above all, to emphasize the significance of the abscess capsule by demonstrating that in the latter is found the important element for safe-guarding brain tissue adjacent to the suppurative lesion, for limiting the infected focus and for providing material for ultimate healing.

EARLIER STAGES IN THE EVOLUTION OF THE CAPSULE IN ABSCESS OF THE BRAIN

It is rather difficult to obtain satisfactory material for the study of the early stages of capsule formation in the so-called contiguous abscess of the brain, i. e., an abscess of the brain secondary to a suppurative process in the middle ear or accessory nasal sinus, since in such an abscess a fatal issue seldom occurs in the very early phase of the suppurative process. In the metastatic form of abscess of the brain which may duplicate almost all of the features of contiguous abscesses, the systemic disease with its primary suppurative focus is the determining factor which may cause death, irrespective of the extent of the cerebral involvement. Under such circumstances the incipient stage in abscess formation, as well as the older phases in capsule organization, may be thus revealed and studied in greater detail; moreover, metastatic

abscesses of more recent origin are often found side by side with somewhat older abscesses and thus one brain may present several phases in the process of abscess formation.

The earliest appearance of an abscess of the brain is that of a small circumscribed area crowded with bacteria, numerous polymorphonuclear

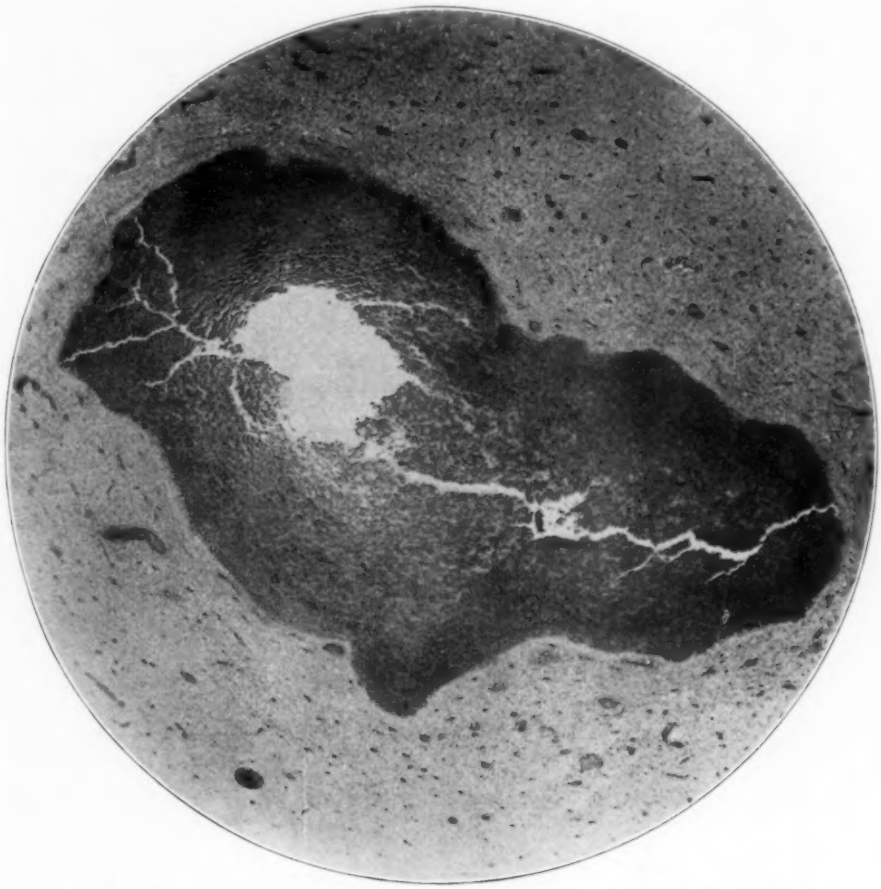

Fig. 1.—Early stage in abscess formation. Note the vascular belt around the suppurative focus. Photomicrograph, × 18.

leukocytes, lymphocytes, red blood cells and disintegrating nerve elements. In the center, a blood vessel may be discerned, the thrombosis and subsequent rupture of which have led to the circumscribed suppuration (fig. 1); more often at this stage there may be found several such areas neighboring one on the other. They very likely have their origin in thrombosis of several branches which are derived from one single

vascular trunk. As the process goes on, the originally separate areas of suppuration join and form a single suppurative process. Often two or more large neighboring vascular trunks may give rise to separate areas of suppuration, but a single abscess cavity may be formed later when intervening tissue becomes necrosed and establishes an intercommunicating channel.

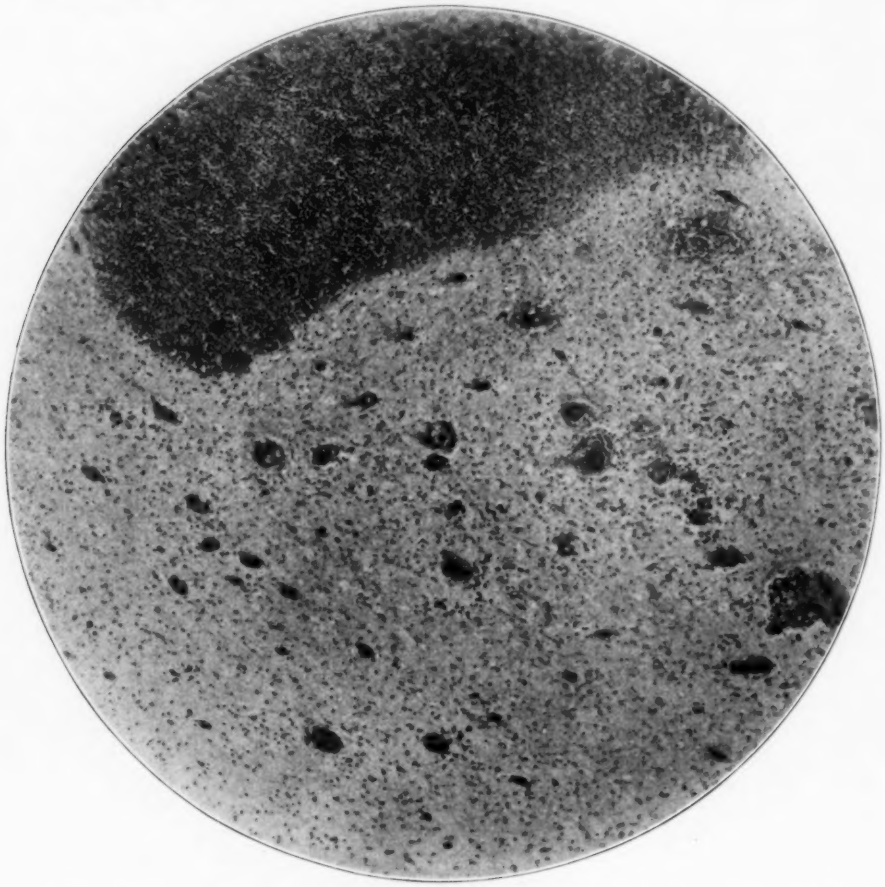

Fig. 2.—Somewhat later stage in abscess formation. Note the wider vascular zone and the lymphocytic infiltration about the individual vessels. Photomicrograph, × 60.

Shortly after the extravasation of the suppurative material from the thrombosed vessel has taken place, reactive phenomena begin to appear at the periphery of the suppurative process. Numerous small blood vessels appear (fig. 2) surrounded by lymphocytic and leukocytic infiltrations. At first the vessels are few in number, forming a well defined

belt around the suppurative zone. Then more vessels come into view, forming several rows; the blood vessels enlarge in size, and are surrounded by denser masses of infiltrating elements. Within a brief period of time lymphocytic elements begin to stray away from the perivascular zones, imparting to the surrounding tissue the character of an interstitial lymphocytic infiltration (fig. 4).

Hassin [3] has shown that these cells, migrating from blood vessels and infiltrating the interstitial tissue around the abscess, are all finally converted into the connective tissue.

Fig. 3.—Gross appearance of metastatic abscess of the brain. Note the multiplicity of the lesion.

Michels and Globus [4] have also shown that lymphocytes leave the blood stream and migrate through the vessel wall into the perivascular space and then into the extravascular territory. It seems reasonable now to assume that the migratory lymphocytes provide the material necessary for the construction of the abscess capsule by their conversion into fibroblasts and ultimately into collagenous structures. In the

3. Hassin, G. B.: Histopathology of Brain Abscess, Arch. Neurol. & Psychiat. **3**:616 (June) 1920; Histopathologic Studies on Brain Abscess, M. Rec. **93**:91, 1918.

4. Michels, N. A., and Globus, J. H.: The So-Called Small Round Cell Infiltrations, Arch. Path. **4**:692 (Nov.) 1927.

course of time the number of vessels in the area, as well as the number of lymphocytes about the abscess, increases progressively. The fibroblasts become more and more numerous and the collagenous tissue denser, with the result that a well organized limiting membrane, consisting of vascular and fibrous layers, is formed (fig. 5).

Beginning with this stage, further alterations in the capsule are best studied in abscesses of contiguous type. Here a fatal issue may

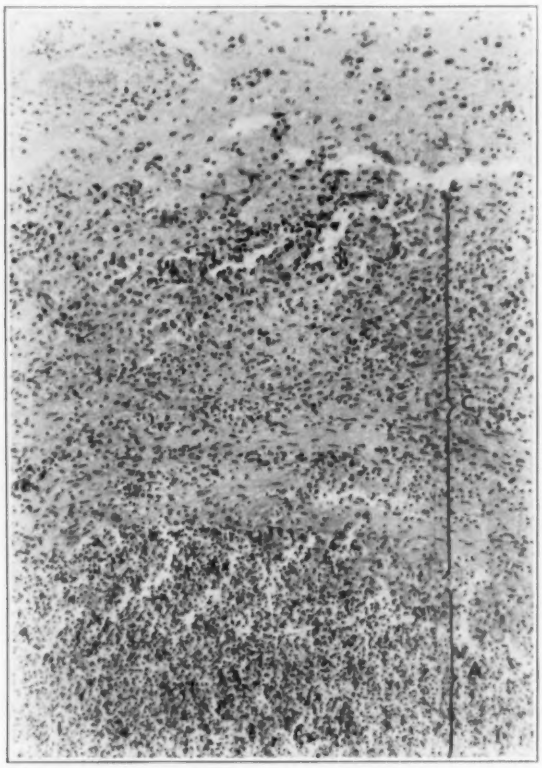

Fig. 4.—Section of the capsule wall (*C*) of an older abscess of the brain (*A*) (see fig. 3). Photomicrograph, reduced from × 150.

often be delayed long enough to permit further development of the capsule, and may, in some instances, lead to almost complete healing of the suppurative process.

As will be seen from a survey of the individual cases herein to be described, the growth of the capsule, which may continue over a period of months or even years, depends on the continuous transformation of the migrated lymphocytes into fibroblasts and mature connective tissue (fig. 6). As the collagenous fibers increase in amount, glial proliferation contributes its share toward the strengthening of the limiting wall about the suppurative process.

RECORDS OF CASES

The fifteen cases to be recorded are best described as instances of contiguous or adjacent abscess of the brain, a term suggested by Eagleton [5] to avoid the unsatisfactory term of primary abscess of the brain. They are either otitic in origin or secondary to infection of the

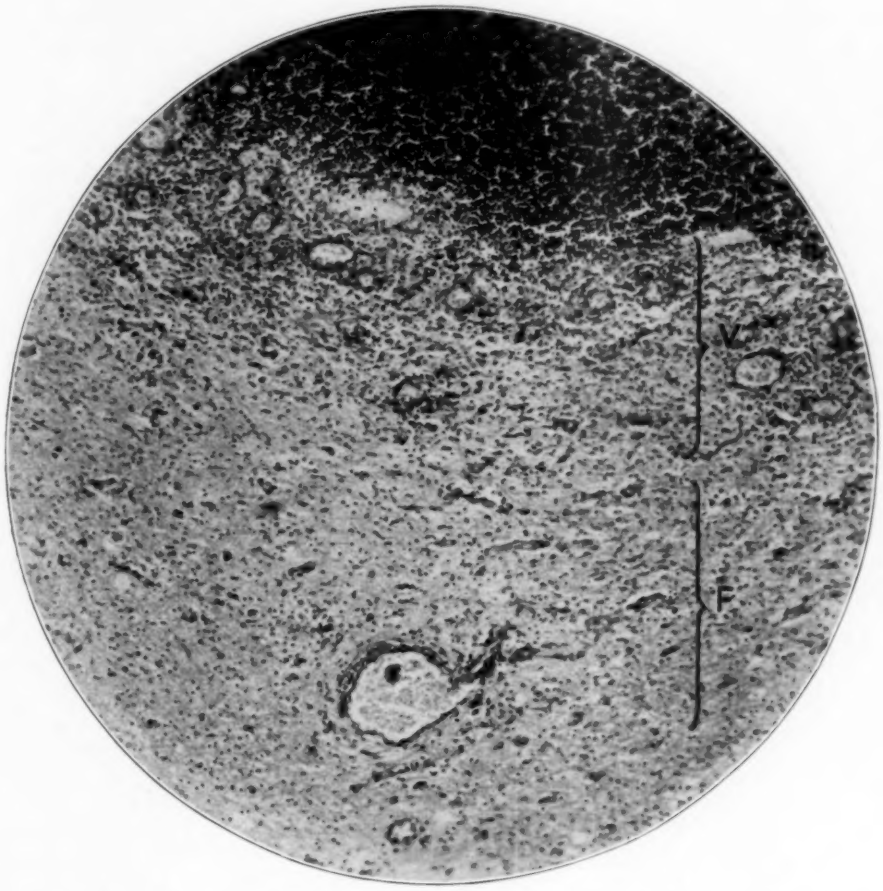

Fig. 5.—Later stage in capsule formation, showing the vascular zone (*V*) and the external fibrous zone (*F*), in metastatic abscess of the brain. The signs of cerebral involvement were of only three days' duration. Photomicrograph, × 75.

accessory nasal sinuses and fall into the following four topographic groups: (1) abscesses of the frontal lobe, (2) abscesses of the temporal lobe, (3) cerebellar abscesses and (4) intrathecal (subdural) abscesses.

5. Eagleton, W. P.: Brain Abscess: Its Surgical Pathology and Operative Technique, New York, The Macmillan Company, 1922.

ABSCESSES OF THE FRONTAL LOBE

CASE 1.—*Recurring respiratory infection, relightening of an infection of the frontal sinus; gradual development of intracranial disease, papilledema, amaurosis, anomia, signs of meningeal irritation and meager localizing signs. No operation. Death. Necropsy.*

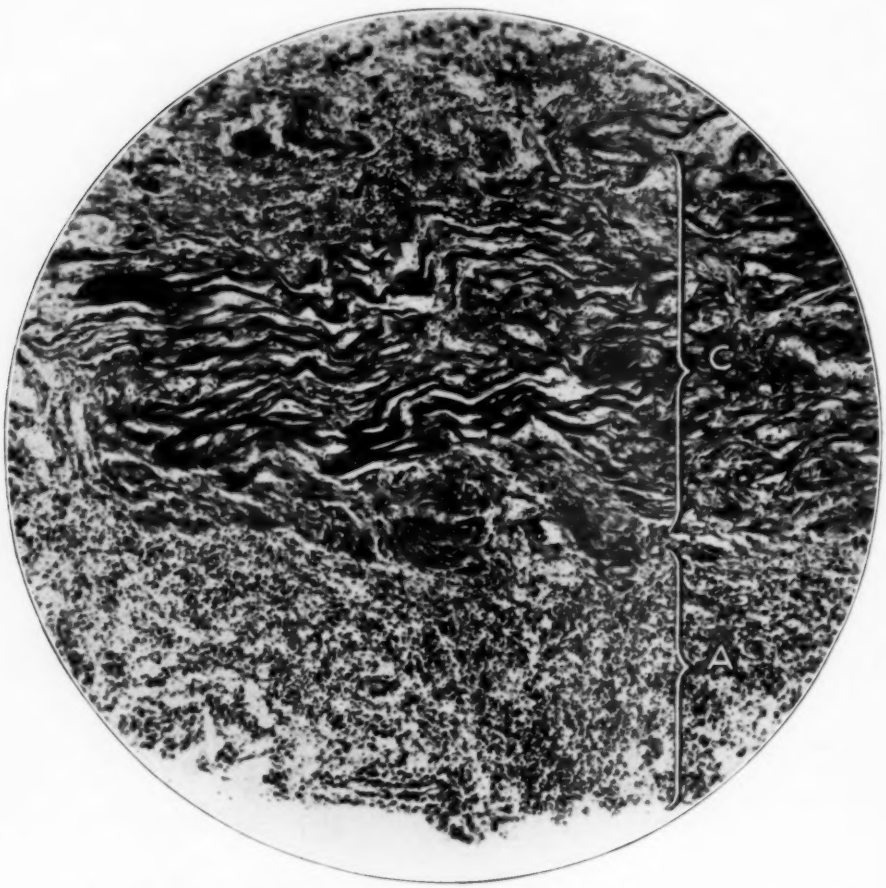

Fig. 6.—Wall of a contiguous abscess of the frontal lobe (*A*), showing the dense fibrous wall of the capsule (*C*). Photomicrograph, × 75.

History.—A. R., a schoolgirl, aged 13 years, had since childhood been subject to respiratory infections. At the age of 2 years she had attacks of pneumonia, and a year later had a recurring attack of some respiratory infection. Later, at various times, she had grip, influenza and a condition diagnosed as an "abscess of the throat." About three months before admission to the hospital, a "cold in the head" again developed. Soon after, she began to complain of impaired vision. This was followed one month later by severe frontal headaches. At this time she had several chills and her temperature rose to 105 F. There were some rigidity

of the neck and a nasal discharge, and the diagnosis of sinusitis was made. Six weeks before admission to the hospital hematuria developed, which lasted a few weeks. Because of the urinary findings the patient was then sent to the Rockefeller Institute where intensive studies of renal disease were being made. There her headaches became more intense and retraction of the head was noted. Drowsiness and dimness of vision continued to progress, and the diagnosis of an inflammatory condition at the base of the brain was made. She was then transferred to Mount Sinai Hospital.

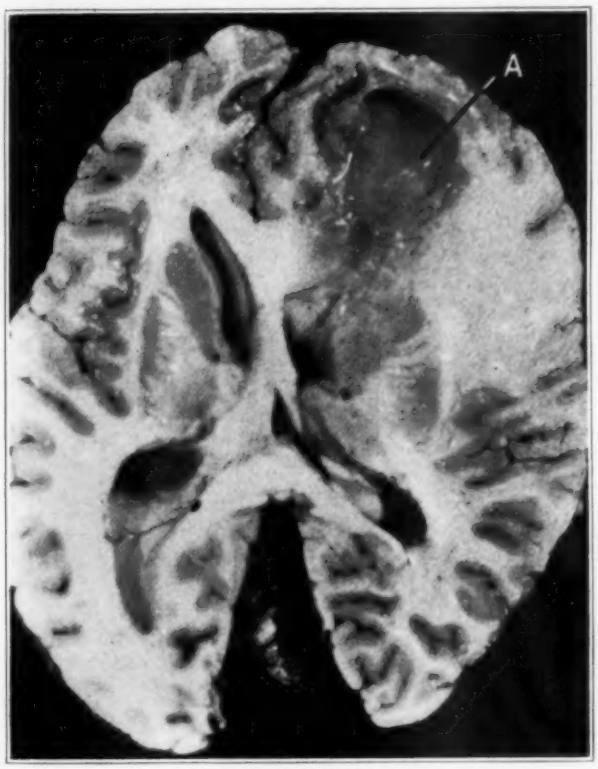

Fig. 7 (case 1).—Gross appearance of abscess of the frontal lobe (*A*).

Examination.—The patient was drowsy and semistuporous, but could be aroused to answer questions. Speech was slow and displayed a bulbar quality. There were bilateral impairment of vision and bilateral papilledema. The pupils were dilated, the right being fixed to light. The left external rectus was weak, and all the deep reflexes were absent. There were moderate retraction of the neck and a right facial weakness.

Course.—Lumbar puncture revealed clear cerebrospinal fluid under normal pressure, containing 8 cells. The patient declined rapidly, passed into a stupor and then coma, and died six days after admission.

Necropsy.—Gross Anatomy: The brain was voluminous and presented features of being under increased intracranial tension; the vessels were somewhat congested. On lifting up the orbital surface of the frontal lobes a purulent exudate was noted

over the cribriform plates. In the region of the gyrus rectus of the right orbital lobe a perforation communicating with a cavity within the brain substance was found. The purulent exudate on the surface of the brain extended back to the sella turcica, where it increased in amount. From there on the entire basal surface of the brain was covered by a similar purulent exudate. On removing the pituitary body there was also noted a perforation of the bony plates forming the roof of the sphenoid sinus. Section of the brain horizontally disclosed a large abscess cavity in the right frontal lobe, measuring about 5 cm. in depth and about 2.5 cm. in diameter (fig. 7). This cavity was fairly well encapsulated and was surrounded by a

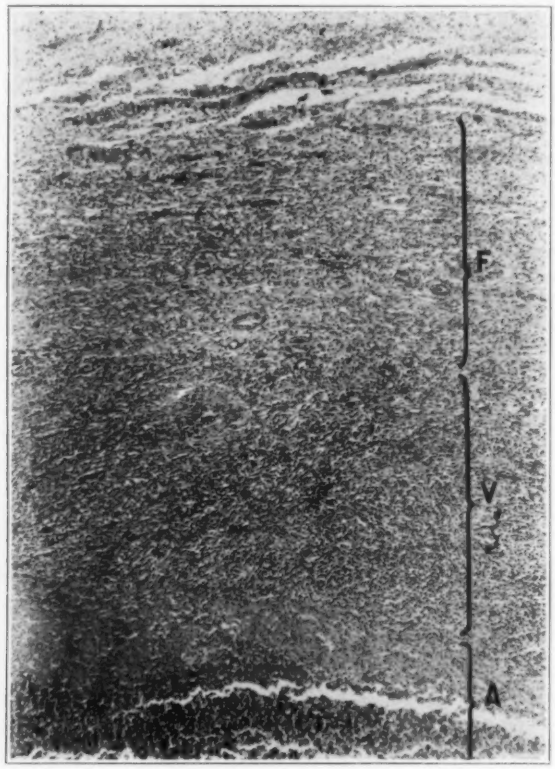

Fig. 8 (case 1).—Section of abscess capsule (see fig. 7), showing its advanced organization. *A*, abscess cavity; *V*, vascular zone; *F*, fibrous zone. Photomicrograph, reduced from × 75.

wide zone of edema in the adjacent tissue of the brain, causing enlargement of the right hemisphere. The lateral ventricles were moderately dilated except for the anterior horn of the right ventricle, which was narrowed.

Microscopic Anatomy: The histologic observations to be reported were limited to the morphologic features noted in the limiting membrane (capsule) of the abscess and adjacent structures. From the microscopic alterations in the capsule it was obvious that the abscess was not of recent origin; its chronicity could be estimated from the thickness of the capsule, the advanced organization and the fibroblastic proliferation in its middle and external layers (figs. 8 and 9).

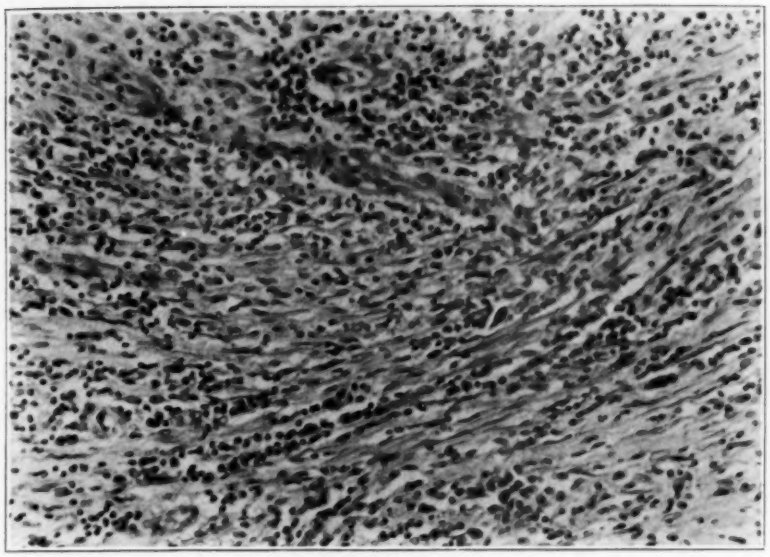

Fig. 9 (case 1).—Fibrous layer in abscess capsule, showing numerous fibroblasts and a large number of blood vessels. Photomicrograph, reduced from × 300.

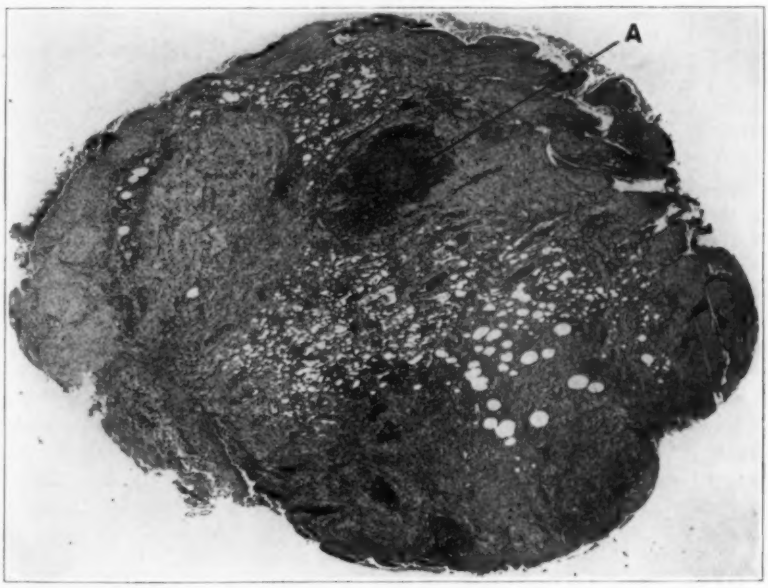

Fig. 10 (case 1).—Abscess stalk (tract). Note the several suppurative foci (*A*), enveloped by dense fibrous material. Photomicrograph, reduced from × 20.

The dependent part of the abscess wall was prolonged into a tract in which more advanced scar formation was revealed alongside of several small suppurative foci (fig. 10). The tract was adherent and partially enveloped by dura, which showed moderate inflammatory changes (fig. 11).

CASE 2.—*Unusual instance of suppurative encephalitis (frontal lobe) associated with panmeningitis and multiple abscess cavities, secondary to disease of the accessory sinuses.*

History.—I. L., a woman, aged 47, was admitted to the Mount Sinai Hospital on four different occasions over a period of two years. Shortly before the first admission (Aug. 9, 1927), while she was nursing a patient with scarlet fever, a bilateral otitis media and edema of the left periorbital region on the left side of the face developed; this was soon followed by edema of the right side of the face

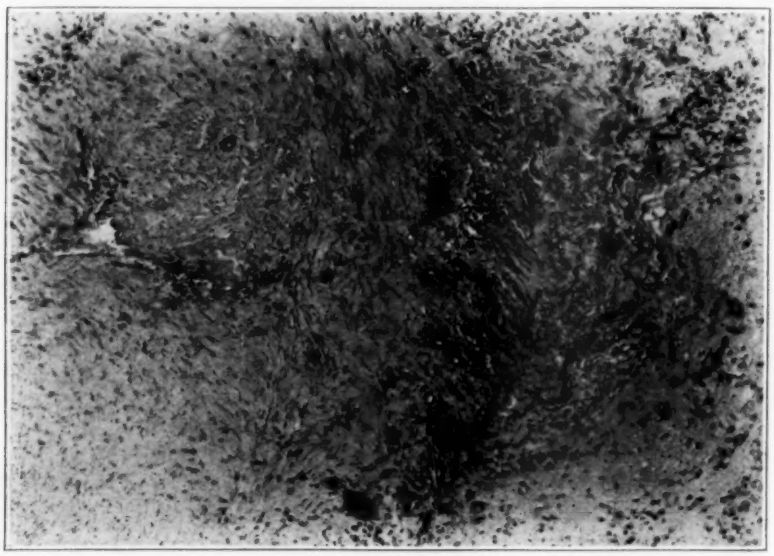

Fig. 11 (case 1).—Section of the dural attachment of the abscess stalk (tract). Photomicrograph, reduced from × 150.

and right eyelid. The left eyelid at first improved, but became swollen again two weeks later. It became red and painful, and finally an abscess in the soft tissues developed. This was opened and drained. The left frontal region continued to be painful and tender. The patient's temperature was elevated, and headache developed. She did not vomit or have chills.

First Admission.—Examination: The left upper eyelid revealed a small area of red swelling which was marked off sharply from the surrounding normal skin. In the center of the swelling there was a small scab but no point of suppuration. The eyelid was tender, as was also the left frontal region. A roentgenogram of the sinuses showed an area of bone destruction in the wall of the left frontal sinus, with a dense shadow in its uppermost portion which was interpreted as a sequestrum. The ethmoid sinuses were cloudy.

Course: A radical left sinus operation was done. Osteomyelitis of the entire anterior wall and floor was found. The patient's condition improved, and at the

end of two months she left the hospital with a discharging fistula in the region of the frontal sinus.

Second Admission.—The patient was readmitted after an interval of two months, complaining of dull pain over the left side of the head and of continued discharge from the wound. No localizing neurologic signs were found at this time. A lumbar puncture revealed normal cerebrospinal fluid containing 3 cells. The patient remained under observation in the hospital for four weeks and then returned home.

Third Admission.—One month later, she was again admitted because of dull pain on the left side of the head accompanied by changes in personality. She was drowsy and apathetic. During the more alert moments she was facetious, and her speech was drawling and monotonous. There was bilateral anomia. The deep reflexes were active with a tendency to bilateral ankle clonus. About ten days after her admission, craniotomy for a probable abscess of the left frontal lobe was performed. No suppurative lesion was found. She remained in the hospital for five weeks more. For one month her mental state continued as before, with episodes of confusion alternating with lucid intervals. During the last week, however, she became mentally clear, but was completely amnesic of the occurrences during the first three weeks of her stay in the hospital. A slight discharge from the wound continued, but it was considered inadvisable to explore the old sinus wound.

Fourth Admission.—For about one year since her last stay in the hospital the patient remained comfortable, with an occasional headache. Occasionally the old supra-orbital scar became tender and sometimes discharged a thin serous fluid. The return of frontal headache and the reappearance of mental changes necessitated a fourth admission to the hospital.

Examination: The gait was slow; the speech was also slow and monotonous. There was a fine tremor in the outstretched fingers. The left pupil was larger than the right and reacted poorly to light. There were slight bilateral external rectus weakness, bilateral papilledema and slight right facial paresis. The left knee jerk and ankle jerk were more active than the right. A left Babinski sign, diminished abdominal reflexes on the left side and impaired hearing in the left ear were revealed. The patient was irritable, facetious and otherwise mentally deteriorated.

Course: The papilledema advanced, and flame-shaped retinal hemorrhages developed. The patient became incontinent. Bilateral pyramidal tract signs appeared with a bilateral Babinski sign. All were more or less marked on the left side. A recurrence of an abscess causing pressure on the right frontal lobe or the formation of a right frontal abscess was suspected. An exploratory craniotomy was done. No abscess was found in either frontal lobe. The patient was reexamined for infection of the accessory sinuses, but none was found. The retinal hemorrhage and papilledema increased. In addition to the definite pyramidal tract signs on the left side, there appeared left hemiparesis and astereognosis in the left hand with normal localization of pain sensibility. This led to the thought that an abscess may have formed in the right parietal lobe, and further exploration was decided on. Exploratory craniotomy disclosed no abscess. Ventriculography was then performed, which showed that the right ventricle was incompletely outlined; it was moderately dilated without gross displacement. The left ventricle was not visualized. The patient did not improve, and three weeks later (September 10) she suddenly complained of pain in the back of the neck. Distinct rigidity of the neck with Kernig signs, more marked on the left side, was found. Aspiration was again done through the previous trephine opening, but no pus was obtained. This procedure gave the patient temporary relief. The papilledema continued to increase. A right subtemporal decompression and needle exploration

were carried out. It was noted that the brain did not pulsate, but there was not much increase in pressure. The temporal lobe seemed indurated. The patient passed into deep stupor and remained in this condition for several weeks following the decompression and then became suddenly alert, talking and answering questions. The papilledema was less marked, but the decompression wound was tense and bulging. The neurologic status remained otherwise unchanged, consisting mainly of right-sided pyramidal tract signs. For the next two weeks the patient's condition varied from day to day. At times she was stuporous; at other times she answered questions fairly well. The following week (November 19) her neck again became rigid. Revision of operation revealed no abscess, but throughout the brain, indurated areas and cavity formations, in addition to a chronic plastic thickening of the meninges, were disclosed. Two weeks later (November 29), aspiration was again done in the region of the right frontal, temporal and parietal lobes, but no abscess was found. The patient failed rapidly after the last procedure and died, seven months after the last admission to the hospital.

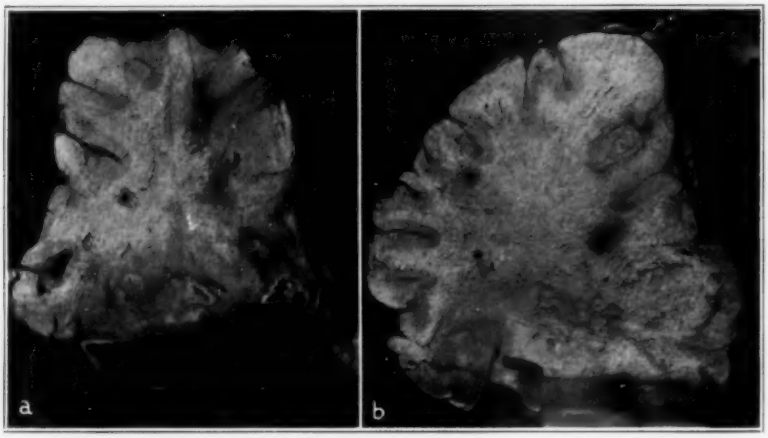

Fig. 12 (case 2).—In *a*, gross appearance of a coronal section of the left frontal lobe. In *b*, gross appearance of a coronal section of the left frontal lobe, somewhat posterior to the section in *a*.

Autopsy.—On reflecting the scalp, defects were found in the skull: one small one in the right parietal region; one large one, about 4 cm. in diameter, in the temporal region; one small one in the frontal region, and one large irregular defect, 4 cm. in transverse diameter, in the frontal bone. All but the frontal defect contained herniating brain tissue.

The brain was somewhat voluminous. There was noted also flattening of the gyri. Over the left frontal lobe near its mesial surface and at the frontal pole the brain substance was adherent to the dura for an area about the size of a silver dollar (fig. 12*a*). The base of the brain was covered by a thick, greenish-yellow purulent exudate. This was particularly marked over the pons and medulla, where the facial and cranial nerves were almost glued down to the underlying structures (fig. 13*c*). The cisterna magna was filled with purulent material. The left frontal lobe displayed on its mesial aspect an indurated area which extended from the frontal pole back as far as the knee of the corpus callosum. It fused the falx cerebri with the adjacent surface of the cerebral hemisphere and thus produced a

very hard scar measuring about 0.5 cm. in thickness (fig. 12*a* and *b*). Throughout the scar tissue there were scattered small areas of yellow discoloration, suggesting caseous necrosis, probably the autolysing centers of abscess formation. There were also areas of greenish-yellow discoloration, variable in size (measuring from about 3 to 5 cm. in diameter), which represented more recent abscess formations. The right frontal lobe in its anterior portion showed a few small hemorrhages in the course of the brain needle employed during the exploratory craniotomy. The ventricles were symmetrical and markedly dilated in all the subdivisions (fig. 13*b*). The third and fourth ventricles were also dilated, while the aqueduct, though not greatly dilated, was still somewhat larger than normal and

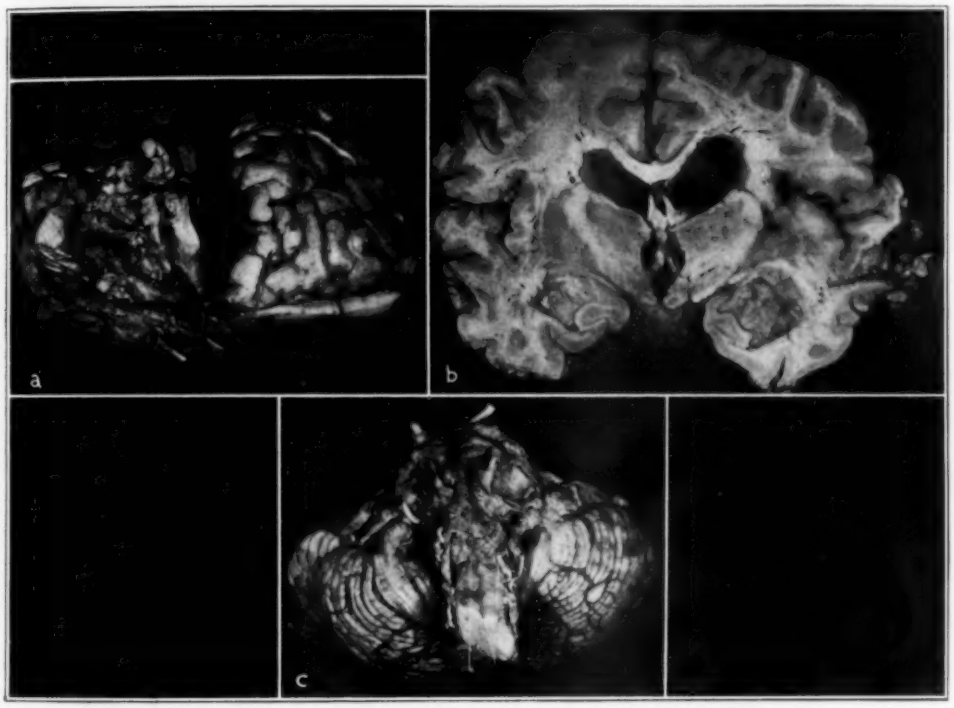

Fig. 13 (case 2).—In *a*, gross appearance of both frontal poles. In *b*, coronal section of brain, showing marked internal hydrocephalus. In *c*, ventral surface of the pons, medulla and cerebellar hemispheres showing marked thickening of the leptomeninges.

rather round in outline. The ependymal lining was moderately thickened and showed many small elevations, presenting a typical picture of chronic granular ependymitis. The choroid plexus was covered with a small amount of thick plastic exudate. The ependymal changes and the alterations in the choroid plexus were most marked on the right side.

The internal hydrocephalus could not be explained by the location and character of the abscess, but in view of the presence of a chronic leptomeningitis and chronic ependymitis, the former indicating the existence of a long-standing obstruction to the normal flow of cerebrospinal fluid, the hydrocephalus must be regarded

as secondary to the chronic leptomeningitis and not to the circumscribed suppurative pachymeningitis.

Microscopic Anatomy: Sections through the involved cerebral hemisphere revealed abscesses in various stages of healing. A small abscess (fig. 14) was found which showed early fibroblastic proliferation in its wall, indicating a fairly recent process. Then again in close proximity an abscess was found in the process of advanced healing, with dense connective tissue forming a strong limiting membrane (fig. 15). In another area, a more densely constructed abscess wall was revealed (fig. 16). This would indicate that repeated reinfections gave rise to

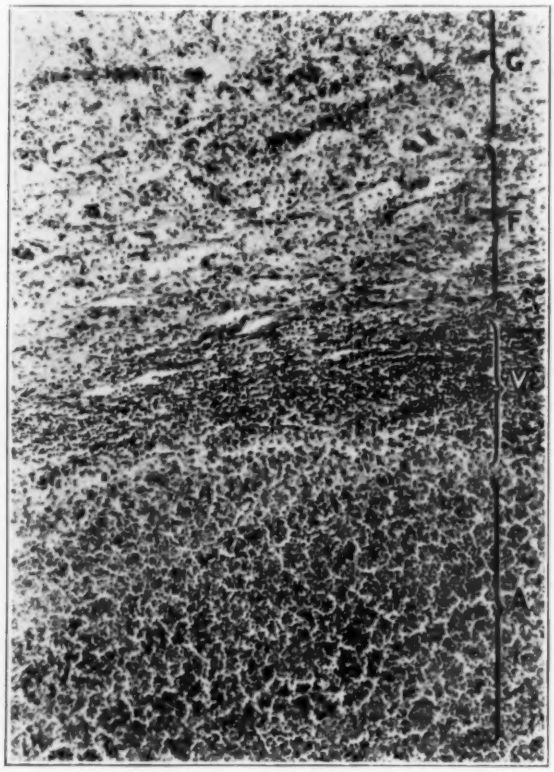

Fig. 14 (case 2).—Section of capsule of a well developed abscess. *A*, abscess cavity; *V*, vascular layer; *F*, fibrous layer; *G*, zone of reactive gliosis. Photomicrograph, reduced from × 125.

newly formed suppurative foci. The surrounding brain tissue showed an inflammatory reaction of the subacute type, infiltration with small round cells and gliosis. The meninges displayed inflammatory changes of acute and chronic forms.

Comment.—This is a rather unusual case. The atypical features manifested themselves from the early onset of the illness and throughout the clinical course. It would appear that from the early onset we were dealing with a multilocular infection in which the bilateral otitis

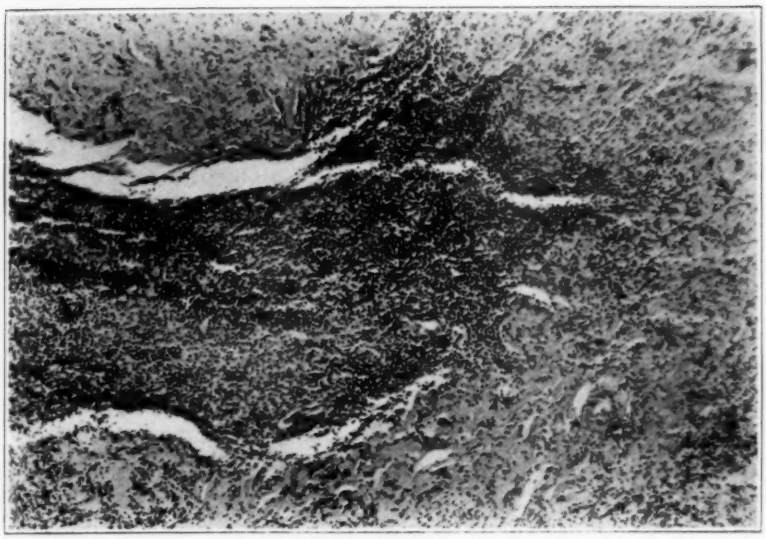

Fig. 15 (case 2).—Section of the wall of a healing abscess. Note the dense fibrous layers about the narrowing suppurative focus. Photomicrograph, reduced from × 125.

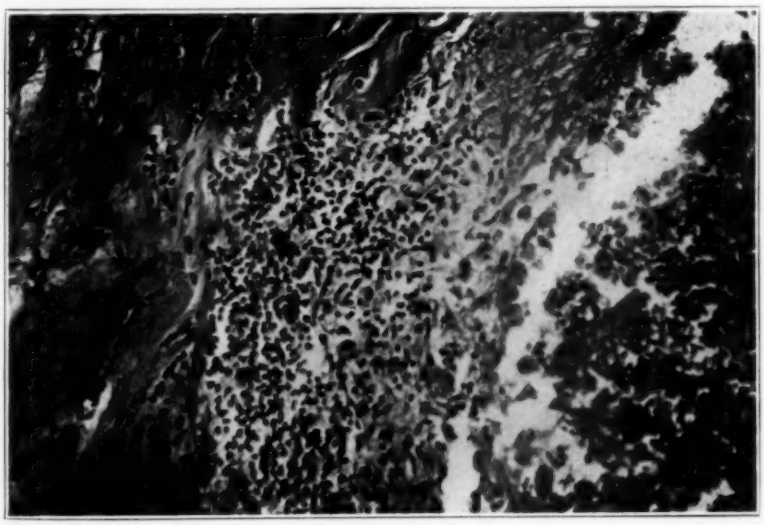

Fig. 16 (case 2).—Section of capsule of a healing abscess. Note the dense character of the collagenous coat. Photomicrograph, reduced from × 240.

media and the left frontal sinus were the most obvious foci of the inflammatory process. It is quite possible that the disease was more in the nature of pansinusitis. At the time of the first operation a well advanced osteomyelitic process in the wall of the left frontal sinus was found. The meninges at that time were apparently spared, as no meningeal signs were noted and no changes in the cerebrospinal fluid were observed though a localized, well encapsulated intracranial abscess may already have been present. The failure of the surgical wound to heal can be accounted for only by the incomplete operative procedure, and thus, on the third admission, evidence of cerebral involvement appeared and a craniotomy in search of the left frontal abscess became imperative. No abscess was found. In spite of that, there was further progress with evolution of new neurologic signs. No attempt at exploration was made at this time.

One year passed without marked changes in the patient's condition, but at the end of this period signs of increased intracranial tension became manifest. They were now associated with involvement of the left third, sixth and seventh cranial nerves and advancing papilledema. What was more striking was the predominance of the symptom pointing to a lesion of the right frontal lobe. It was thought that the abscess of the left frontal lobe, by indirect pressure on the right hemisphere, was responsible for this, and a revision of the left craniotomy field was done. No abscess was found. There was further advance in the signs of increased intracranial tension. An exploratory craniotomy was performed on the right side, but no pus was found. Ventriculography disclosed no shifting of the ventricles but moderate ventricular dilatation. Shortly after, meningeal signs appeared. Another exploratory craniotomy on the left side was done. No frank abscess of the brain was found, but it was observed that the brain did not pulsate and appeared to be retracted. This was interpreted as indicating some secondary degenerative process. A final attempt was made in search of a localized degenerative lesion, but a fairly diffuse indurated process associated with marked thickening of the meninges and focal cavity formation were found. In reconstructing the clinical picture in the light of the anatomic findings, it would seem that from the very early stages of the clinical course a pachymeningitis was present. This extended to form a circumscribed panmeningitis, and was followed by a slow spread of the leptomeningitic process along the base of the brain. It is the latter that caused the obstruction to the normal flow of cerebrospinal fluid and the communicating form of hydrocephalus which was responsible for the signs of increased intracranial tension. Synchronously with this process there occurred a chronic productive suppurative encephalitis and a basilar meningitis which led to the meningeal signs

and the cranial nerve palsies. It is possible that a more thorough exploration of the left frontal sinus at the very first operation would have arrested the course of events which led to the fatal termination.

CASE 3.—*Ethmoiditis. Ethmoidectomy followed by apparent recovery. Four months later, symptoms of intracranial involvement with signs of an abscess of the brain. Craniotomy. Abscess found and drained. Improvement for ten days. Abscess reentered and drained. Temporary relief. Patient discharged from the hospital. Readmitted one week later with symptoms of recurrent abscess of the brain. Abscess drained again. Meningitis. Death. Necropsy.*

History.—S. H., aged 45, was admitted to the hospital twice. Six months prior to his first admission he was operated on for an ethmoiditis. He made an apparently good recovery. Four months later, however, he began to complain of headache and dizziness. He vomited repeatedly. His condition became alarming, and he was brought to the hospital.

Examination.—The patient was in a semistupor. His respiration was Cheyne-Stokes in type. There was moderate discharge from his ears, the neck was rigid, and there were a mild bilateral Kernig sign, bilateral papilledema and flatness of the left side of the face.

Course.—A lumbar puncture revealed clear, colorless cerebrospinal fluid under moderately increased pressure and containing 9 cells. The rapidly increasing papilledema (4 diopters in the right eye and 3.5 diopters in the left) demanded surgical intervention, and an exploration for an abscess of the right frontal lobe was decided on. The localizing signs were hemihypalgesia on the left side, weakness of the rectus externus on convergence, weakness of the left side of the face, ptosis of the left upper extremity and mental changes. The finger-to-nose test revealed tremor of the right upper extremity; both upper abdominal reflexes were diminished; the lower left was less active than the right; there was weakness of the left grasp. Vision for small objects was limited, especially in the left eye. Craniotomy was performed. A deep subcortical abscess at a depth of 5 cm. was found, and pus was evacuated. Gram-positive cocci were found (Staphylococcus aureus). Following the operation the patient's condition seemed to improve for a period of ten days, although the fundus remained unchanged. At the end of this time he again complained of severe frontal headache; he was drowsy, and his breathing became of a Cheyne-Stokes character. The abscess was again drained. The patient again improved steadily, but the papilledema advanced with retinal hemorrhages, and dimness of vision continued to persist. This led to the belief that there were other foci present. The patient's condition, however, improved sufficiently to permit his return home. One week after his discharge from the hospital he was again admitted with a recurrence of headache, vertigo, dimness of vision and drowsiness. The fundi now showed papilledema (4 diopters in the right eye and 5 diopters in the left), with fresh hemorrhages. A lumbar puncture yielded clear cerebrospinal fluid under normal pressure. The abscess was reentered through a right temporal opening and the pus drained. Following the operation the patient's condition became gradually worse. Marked rigidity of the neck and bilateral Kernig and Brudzinski signs developed. The deep reflexes were diminished, with slight preponderance on the left side. A second lumbar puncture yielded 25 cc. of cloudy cerebrospinal fluid under pressure, with a cell count of 335. The patient died five days after this second admission.

Necropsy.—Gross Anatomy: On removal of the calvarium, the dura was found to be adherent in two regions; first, at the point of the operative field, and second, over the cribriform plate. The pia-arachnoid was somewhat thickened, but

presented no evidence of either serous or purulent exudate. The brain showed marked flattening of the gyri and diminished consistency, particularly over the left frontal region. On exposing this region, a rubber tube inserted into the brain substance for drainage purposes was recovered. This opening was found to communicate with a large abscess cavity (fig. 17). On removal of the brain, the orbital surface of the right frontal lobe was found adherent to the dura and the underlying bony structure. It was necessary to cut the brain at the point of adhesion to remove it. Further study of the structures in the region of the cribriform plate revealed that this portion of the ethmoid bone was absent. A

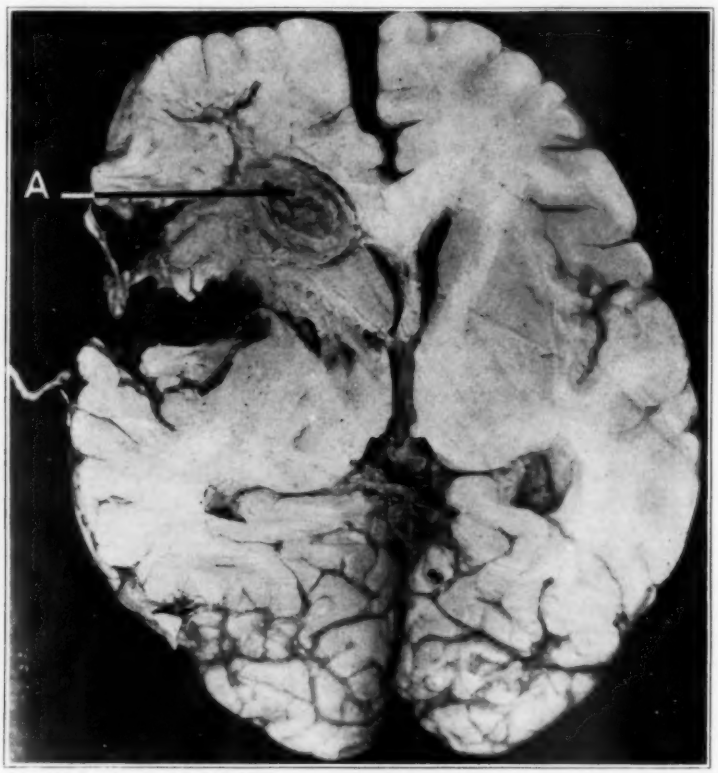

Fig. 17 (case 3).—Gross appearance and location of healing abscess (*A*).

large opening was found communicating with the nasal cavity. The under surface of the brain, close to the base of the right olfactory nerve, showed a small puncture opening communicating with a rather small abscess in the right frontal lobe.

Microscopic Anatomy: The capsule in this case, when examined even with the unaided eye, showed advanced thickening of the abscess wall. Microscopic preparations revealed that robust bundles of collagenous fibers had replaced the young fibroblasts and the small capillaries (fig. 18). Small collections of leukocytic elements were seen in the lumen of the abscess, and a negligible number of such cells were noted in between the fibers. The abscess was unquestionably on its way to complete healing; one could no longer distinguish the component layers of the capsule.

Comment.—The two essential points in this case may be summed up by saying: 1. The abscess was unquestionably the result of a necrotic process in the roof of the ethmoid bone and formed by contiguity. 2. The clinical history and the histologic observations point to chronicity of the process.

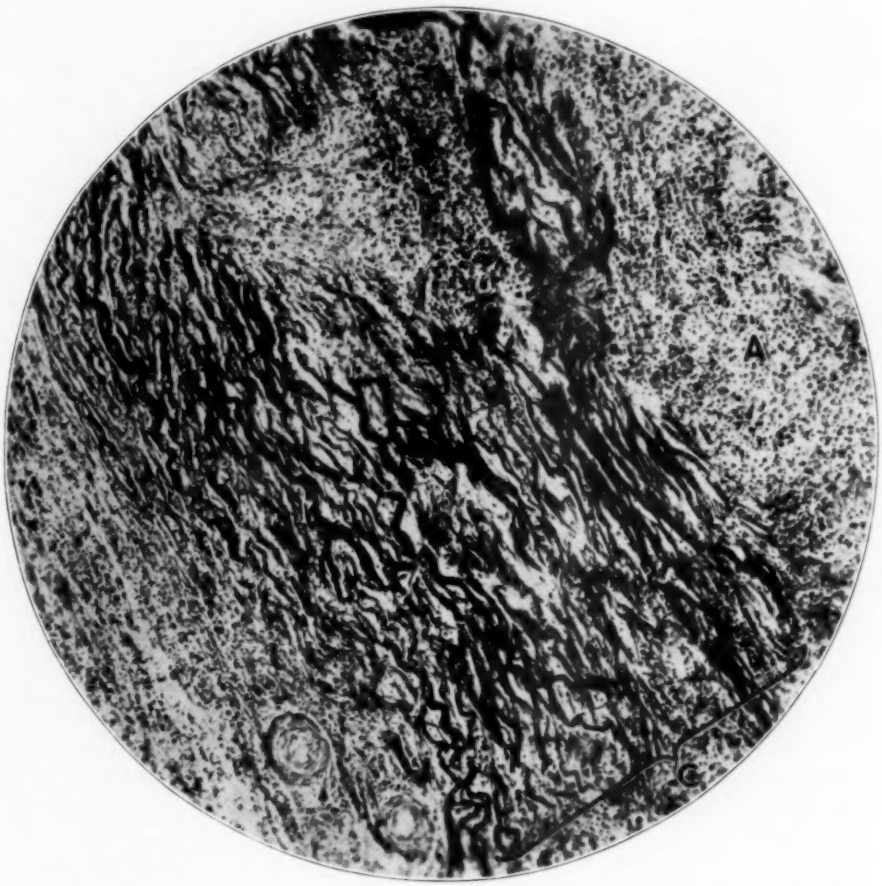

Fig. 18 (case 3).—Section of abscess capsule. *A*, abscess cavity; *C*, capsule. Photomicrograph, × 75.

This case is a good illustration of the inherent healing properties of the capsule. As seen from the gross and microscopic preparations, the abscess cavity was surrounded by an exceedingly thick wall protecting the adjacent tissue from the spread of the infection. It would seem that were the abscess treated as a tumor and removed *in toto*, better results would have been obtained, for attempt at drainage could

not be successful because of the thickness of the capsule. It is not unlikely that this surgical procedure only helped to spread the infection.

The indications in similar cases are first to locate the lesion occasionally by an open exploration and then remove it in its entirety, and not attempt to drain it.

CASE 4.—*Infection of the frontal and ethmoid sinuses, followed by a jacksonian attack. Diagnosis of abscess of the right frontal lobe made. Exploratory craniotomy performed. No abscess found. Death. Necropsy.*

History.—E. G., a woman, aged 30, was operated on for infection of the frontal and ethmoid sinuses one year prior to her admission to the hospital. There remained a discharging fistula in the right brow, which continued to drain for about eight months. At the end of this period a sequestrum was removed and the fistula closed. Two months later the patient suddenly lost consciousness and generalized convulsions developed. The convulsive seizures, lasting three or four minutes at a time, recurred from fifteen to twenty times during that day. The next day the patient regained consciousness and, except for the headaches and persistent nasal discharge, remained apparently well until the night of admission. At this time a sudden change in her mental condition was noted. She stared blankly into space, did not answer questions and was indifferent to the surroundings. Soon after, convulsive seizures again set in. These were jacksonian in character and began in the left hand, spreading to the face and left leg; they recurred every twenty minutes. The patient vomited several times.

Examination.—The pupils were equal, but did not react to light. The eyes were in conjugate deviation to the right; there were bilateral corneal anesthesia and a complete hemiplegia involving the left side of the face and left extremities. A left Babinski sign was elicited. The left knee jerk and ankle jerk were more active than the right. There was bilateral papilledema of about 1 diopter. A lumbar puncture yielded clear, cerebrospinal fluid containing 6 cells.

Course.—While in the hospital the patient passed through several jacksonian attacks. These convulsive seizures would begin in the left arm and spread to the left side of the face and left leg, with the eyes rotating to the left during such attacks. A diagnosis of abscess in the right frontal lobe was made, and an exploratory transfrontal craniotomy was performed. The brain was aspirated in various directions, but no abscess could be located. Following this operation the jacksonian attacks became more frequent, and the patient's temperature rose gradually to 107 F. She died two days after admission.

Necropsy.—Gross Anatomy: The dura was normal. The pia-arachnoid was free from adhesions, thickening or exudate. The brain substance in the region of the frontal pole, about 1 cm. posterior to the anterior margin, showed reduced consistency and some blanching. An incision made at about 3 cm. from the anterior pole disclosed a small abscess surrounded by a thick and indurated wall (fig. 19). The abscess with its capsule measured about 2 cm. in diameter, and an incision showed a markedly but irregularly thickened, fibrosed capsule. The abscess cavity itself was small, measuring only 0.5 cm. in diameter, and contained a small amount of grayish-white exudate. The needle puncture was found at a distance of 2 cm. from the abscess cavity, extending backward and entering the corpus caudatum.

Microscopic Anatomy: Sections through the abscess cavity and its wall showed an extremely thick capsule composed of dense collagenous fibers, encircling an extremely small area of vascular tissue (fig. 20). The brain tissue directly adja-

Fig. 19 (case 4).—Gross appearance of healed abscess (*A*).

Fig. 20 (case 4).—Fibrous scar, marking the place of the healed abscess. Photomicrograph, × 100.

cent to the abscess wall was edematous and showed marked glial proliferation (fig. 21). It was highly vascular and showed many infiltrated blood vessels (fig. 22). The infiltrations consisted of lymphocytes and gitter cells. A few small hemorrhages were seen in the direct vicinity of the abscess. Other sections in remote parts of the brain showed nothing except a few vessels infiltrated by an occasional lymphocyte or gitter cell filled with pigment. There were no other changes.

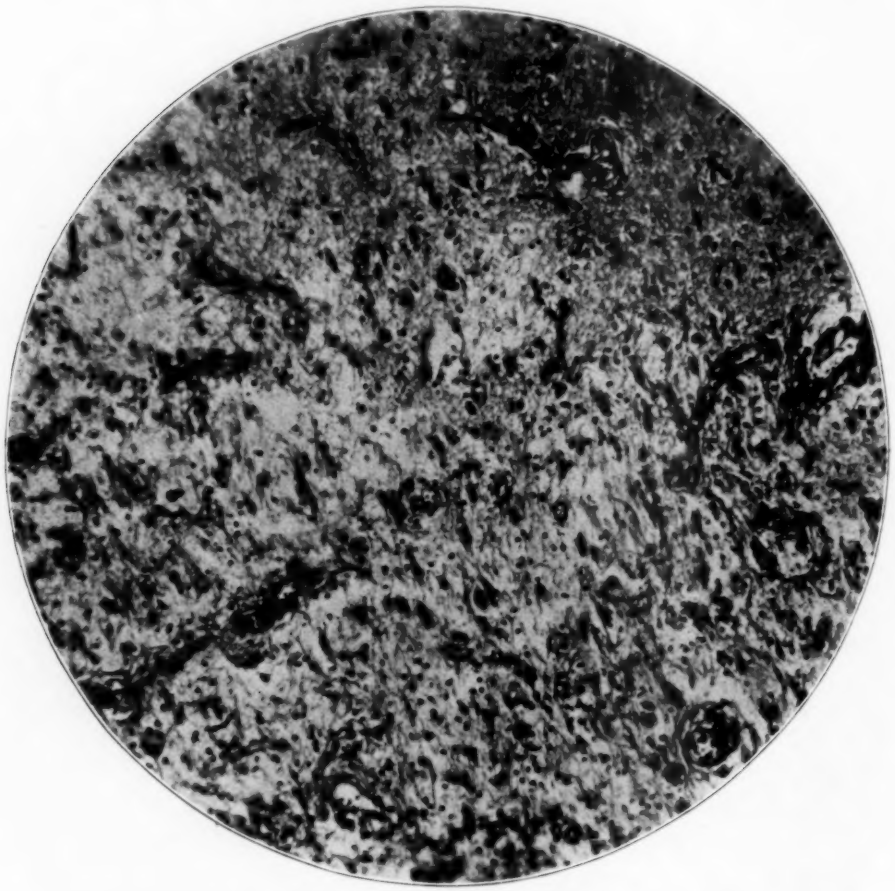

Fig. 21 (case 4).—Glial proliferation in brain tissue adjacent to wall of abscess. Photomicrograph, × 130.

Comment.—This case offered no great diagnostic difficulties. The history of a sinus infection, the performance of an ethmoidectomy, followed in course of time by manifestations of a focal lesion in the brain, could lead to no other diagnosis but abscess of the brain. The objective neurologic findings, including the jacksonian attacks and the conjugate deviation of the eyes, pointed to the frontal lobe as the seat of the

lesion. The mental picture could also be regarded as localizing the lesion in the frontal region. However, the lapse of time between the acute sinus infection and the development of the signs of increased intracranial tension should have been regarded as evidence that the abscess was of old standing and very likely well encapsulated. Under such circumstances, ordinary methods of approach by means of an aspirating needle could not promise much. Successful detection of the expanding lesion in such instances is possible only by means of wide exposure and open exploration. Then the abscess could have been removed *in toto* in the manner in which neoplasms are treated. This view is justified by the histologic observations in this case. They indicate that here the abscess provoked signs and symptoms, not as an inflammatory process, but as an expanding lesion, and hence should have been treated as such. This case also illustrates the great potency of the limiting membrane for healing and cicatrization. It is rather striking that abscesses of the frontal lobe are those in which the healing process may progress for a long time and reach a high degree of cicatrization. This is readily explained by anatomic and physiologic factors and in the light of experience with tumors of the frontal lobe. The frontal lobe, and particularly the right frontal lobe, may often lodge massive expanding lesions without giving rise to any disturbance in function or provoking manifestations of increased intracranial tension. The latter is due to the fact that in this position a tumor or abscess does not disturb the normal flow of the cerebrospinal fluid. Hence hydrocephalus develops late, if at all, with the result that the signs and symptoms of increased intracranial tension are late in development. Then again, since the right frontal lobe contains fewer centers of importance, the local disturbances are also less pronounced than with lesions in other parts of the brain.

ABSCESSES OF THE TEMPORAL LOBE

CASE 5.—*Chronic purulent otitis media of many years' standing. Relightening of the process with the development of signs of increased intracranial tension. Focalizing cerebral signs in the left temporal lobe. Meningeal signs. No operative intervention. Death. Necropsy.*

History.—M. H., aged 47, had had moderate deafness due to chronic disease of the middle ear since childhood. For the past three years he had had pain in the left side of his face and head.

Examination.—The patient was drowsy, but when aroused appeared dull and slow. Both ears showed perforated drums. There was a profuse purulent discharge from the left ear. Hearing was impaired in both ears, but more so in the right. Caloric stimulation revealed active labyrinths. The abdominal and cremasteric reflexes were absent. No other neurologic findings were elicited.

Course.—On the day following the patient's admission his temperature, which was normal on the previous day, rose to 104 F. A temporal anomia, left facial weakness and a Babinski sign on the right side developed. The diagnosis of an abscess of the left temporal lobe was made. A blood count showed 14,300 white cells with 60 per cent polymorphonuclear leukocytes, 30 per cent lymphocytes, 2 per cent eosinophils and 1 per cent monomorphonuclears. The pulse rate varied

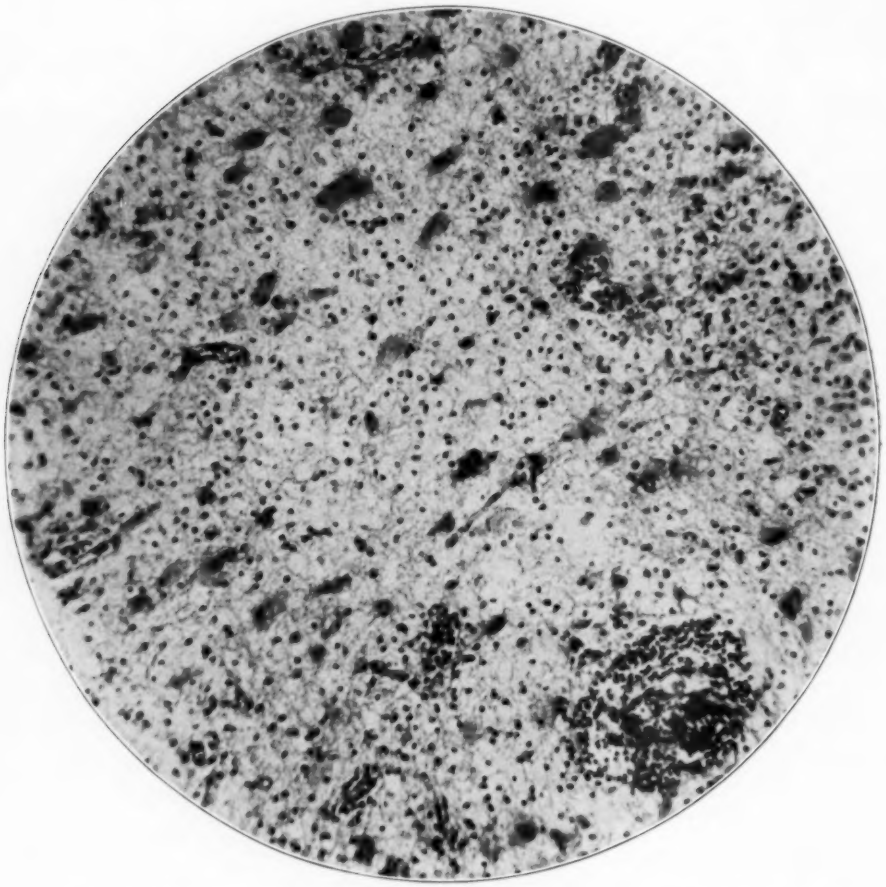

Fig. 22 (case 4).—Few vessels showing lymphocytic infiltration and gliosis in region adjacent to abscess capsule. Photomicrograph, × 140.

between 50 and 80. The patient became drowsy and the aphasia persisted. Four days after admission the neck became rigid.

A lumbar puncture revealed cerebrospinal fluid under increased pressure containing 5,000 cells with 90 per cent polymorphonuclear leukocytes. A culture revealed streptococci. The patient's condition, after a brief period of improvement, declined steadily. He died on the sixth day without operative intervention.

Necropsy.—The left mastoid cells were soft and necrotic. There was a widespread patchy purulent meningitis throughout the surface of the brain. It was more

marked at the base. At the posterior part of the middle temporal convolution there was an abscess about 5 cm. in diameter (fig. 23). It was rather superficial and contained a moderate amount of thick yellowish pus.

Microscopic Anatomy: A fairly definite capsule, though not discernible grossly, was recognized in histologic study of the abscess wall (fig. 24). Its recent origin was also detected by scant fibroblastic proliferation. The discrete separation from the brain tissue is quite characteristic of the development of an abscess when not interrupted by some extrinsic event.

Comment.—In this case we are dealing with a rather typical instance of an abscess of the temporal lobe occurring in a person with a history of chronic disease of the middle ear. In view of the fact that a high cell count and gram-positive organisms were found in the cerebrospinal

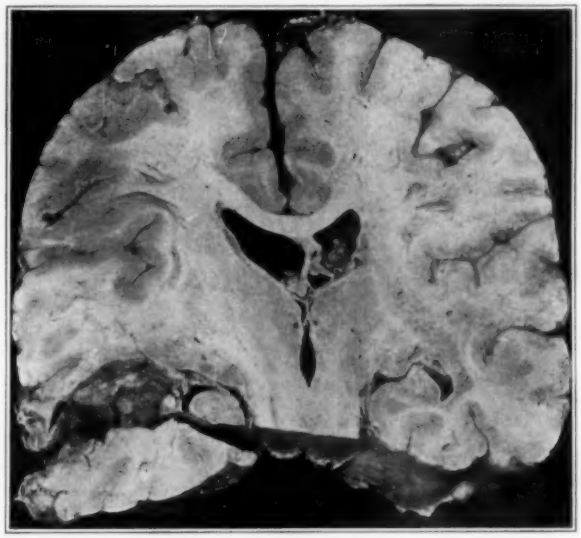

Fig. 23 (case 5).—Gross appearance and location of abscess.

fluid, no operative procedure was attempted, despite the fact that a diagnosis of abscess of the brain was made.

CASE 6.—*Acute otitis media of the left ear, four weeks before admission; incised and drained. Discharge stopped at the end of a week and then the patient appeared to be well for ten days. Signs of increased intracranial tension appeared: Craniotomy and aspiration of abscess of the left temporal lobe. Temporary improvement. Headache returned and temperature rose to 106 F. Rapid decline. Death. Necropsy.*

History.—H. R., a youth, aged 15, was operated on for an acute otitis media of the left ear four weeks before admission. The incised ear continued to drain for a while, but a week later the discharge stopped and the patient was free from symptoms for about ten days. He then began to complain of pain in the head, which gradually increased in intensity and was followed by drowsiness. The patient was then brought to the hospital.

Examination.—The patient was drowsy and difficult to arouse. He had severe headache. Examination showed slight rigidity of the neck, a bilateral Kernig sign, more marked on the right side, right central facial weakness and a sensory aphasia. The right pupil was larger than the left, both reacted well to light and in accommodation. There was a right visual field defect in the form of an incomplete homonymous hemianopia. The pulse rate was slow, varying between 48 and 80. The right knee jerks were less active than the left.

Course.—Craniotomy was performed under local anesthesia. A button of bone was removed from behind the ear. About 3 ounces (88 Gm.) of thin pus was

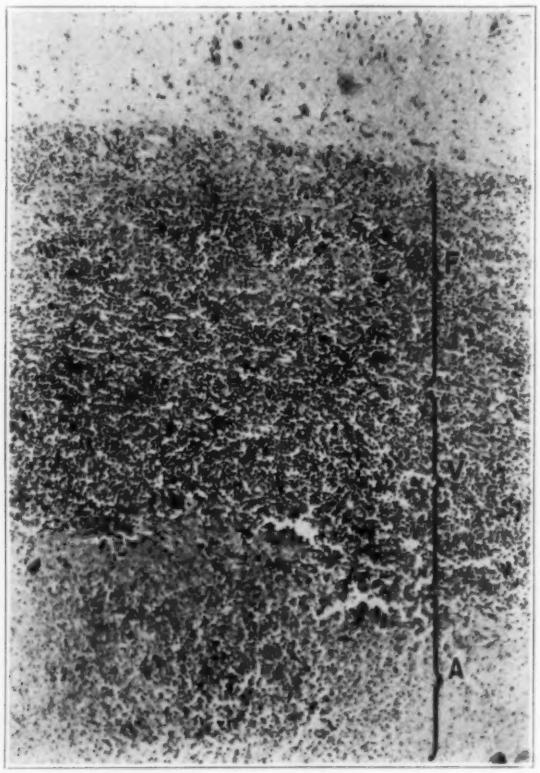

Fig. 24 (case 5).—Section of capsule of abscess. *A*, abscess cavity; *V*, vascular zone; *F*, fibrous zone. Photomicrograph, reduced from × 115.

removed. The following morning the pulse became slow. The dressing was changed; as there was no drainage from the tube the cavity was irrigated and the detritus and clotted blood were washed out. There was no change in the general condition for two days; then the child became restless and finally stuporous. The right pupil became fixed to light; the left was contracted, but reacted to light. The pulse became weaker and breathing labored, and the patient died.

Necropsy.—The dura did not show any gross alterations except for a slight hemorrhagic discoloration surrounding the drainage tube. The latter, inserted in the second temporal convolution posteriorly, was surrounded by an irregular opening of softened necrotic brain substance. The left hemisphere, especially the

temporoparietal area, was much softened. The mesial surface of the left temporal lobe corresponding to the hippocampal region was marked by a small area of discoloration. The leptomeninges in the sylvian fissure and at the base of the brain showed thickening and opacity. The left cerebellopontile angle was filled with an accumulation of purulent fluid. On section, a large somewhat collapsed abscess cavity was found in the left temporo-occipital region involving the posterior one third of the temporal and the anterior one third of the occipital lobe. The ventral aspect of the abscesses reached the basal surface of the hemisphere and was

Fig. 25 (case 6).—Gross appearance and location of abscess.

separated from the meningeal lining by an exceedingly narrow zone of brain tissue (fig. 25). The interior of the abscess had a thick capsule, about 3 mm. in width. It was well organized, resisting the cutting knife and was surrounded by a hemorrhagic zone irregular in outline. An area of softening, greenish-yellow in color, was found directly around the hemorrhagic zone spreading for a distance of 1 cm. or more into the white substance of the temporoparietal region.

Microscopic Anatomy: The gross appearance of the abscess did not reveal its true structure. Its vascularity gave the appearance of an extensive hemorrhage. But, on microscopic observation, it was noted that a true capsule was in the process

of formation (fig. 26) and that this capsule already showed in its wall well defined fibroblastic proliferation (fig. 27).

Comment.—In this case the localization of the lesion did not present a serious problem. It is more difficult to explain the rapid fatal issue and the hemorrhagic condition in the wall of the abscess unless it was the result of trauma caused by the drainage tube. Of course, the fairly diffuse meningeal process may be considered as the cause of death.

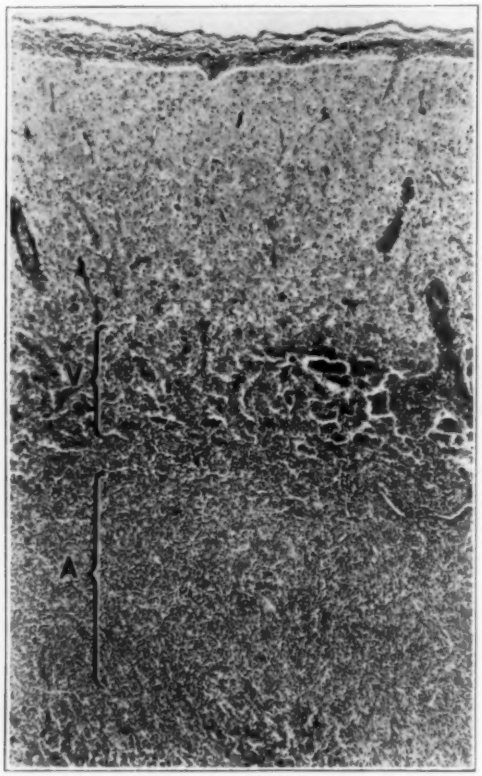

Fig. 26 (case 6).—Section of abscess capsule. *A*, abscess cavity; *V*, vascular zone. Photomicrograph, × 115.

CASE 7.—*Chronic discharge from the ear of two years' standing: Acute mastoiditis. Mastoidectomy. Subdural abscess found. Temporary improvement. Return of symptoms. Exploratory craniotomy. Incomplete drainage of abscess. Meningitis. Death. Necropsy.*

History.—I. M., a boy, aged 9 years, had a discharging right ear for two years. Three days before his admission to the hospital pain suddenly developed in that ear. On the day of admission he had a chill. His temperature rose. He became drowsy and vomited once.

Examination.—The child was acutely ill. He was in a deep stupor and vomited repeatedly. The left pupil was larger than the right. The neck was rigid, there were marked Brudzinski, bilateral Kernig and Babinski signs and the abdominal reflexes were absent. A lumbar puncture revealed cloudy cerebrospinal fluid under increased pressure, containing 3,520 cells, all of which were polymorphonuclear leukocytes. Smears showed no organisms. The blood count showed 22,500 white cells, of which 85 per cent were polymorphonuclear leukocytes and 15 per cent lymphocytes.

Course.—Three days after admission a right mastoidectomy was done and thick foul pus was obtained. The sinus was exposed and many granulations were found. On exploring the floor of the middle fossa an epidural abscess was disclosed. On further exposure the dura was found covered with granulations, and on slight manipulation foul pus exuded from the temporal lobe; about 5 cc. of it

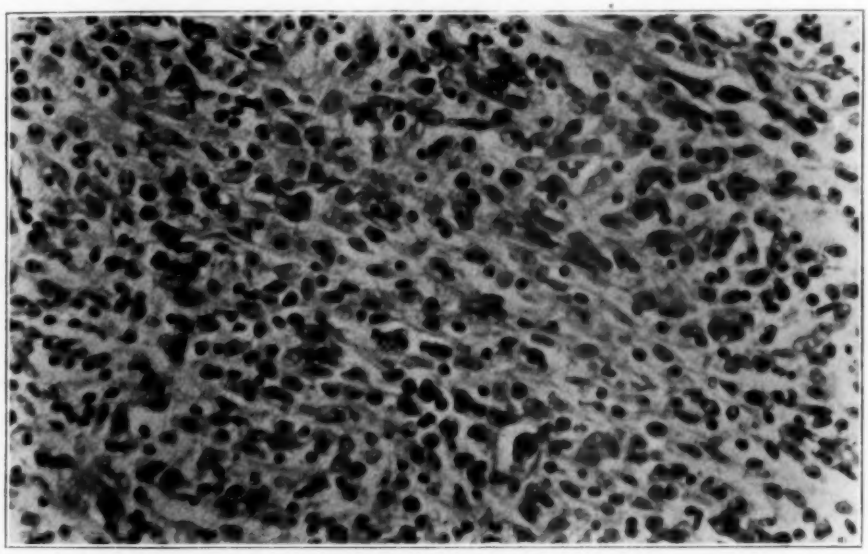

Fig. 27 (case 6).—Fibroblasts in the periphery of the vascular zone. Photomicrograph, × 300.

was obtained. The patient seemed to improve for four days. However, on the fifth day after the operation the temperature rose, and severe pain developed in the supra-orbital region. The Brudzinski sign and a right Kernig and bilateral Babinski sign were again elicited. The neck became rigid and the patient became irrational. A larger tube was inserted into the abscess cavity, lumbar punctures were frequently done and the condition of the patient began to improve again. Four days later the patient became drowsy and a left hemiparesis developed. The drainage tube was removed, and pus was recovered under tension. Lumbar punctures were continued twice daily and now, in addition to the increasing cell count, organisms were recovered. It was thought that the clinical picture was now that of a bacterial meningitis, and surgical intervention was regarded as contra-indicated. The continuation of subarachnoid drainage by repeated lumbar punctures was thought to be the measure of choice. The patient declined steadily, and died four weeks after admission to the hospital.

Necropsy.—At the site of the surgical wound the pia-arachnoid and dura were adherent to the surgical opening in the skull as well as to the underlying brain tissue forming a canal lined by granulation tissue connecting the abscess with the surgical wound. The pia-arachnoid, at the base and at the left dorsolateral surface, showed an accumulation of yellowish pus in the cisterna as well as in the sulci. On palpating the right hemisphere a marked sagging of a large area in the temporal lobe was noted. The rest of the brain showed marked flattening of the gyri. On incision a large abscess cavity filled with yellowish pus was found in the temporal lobe (fig. 28), involving an area about 6 cm. in length and 4 cm. in depth.

Microscopic Examination: In this case the gross appearance of the capsule is in full accord with the histologic features. A well marked fibrous layer, rich in

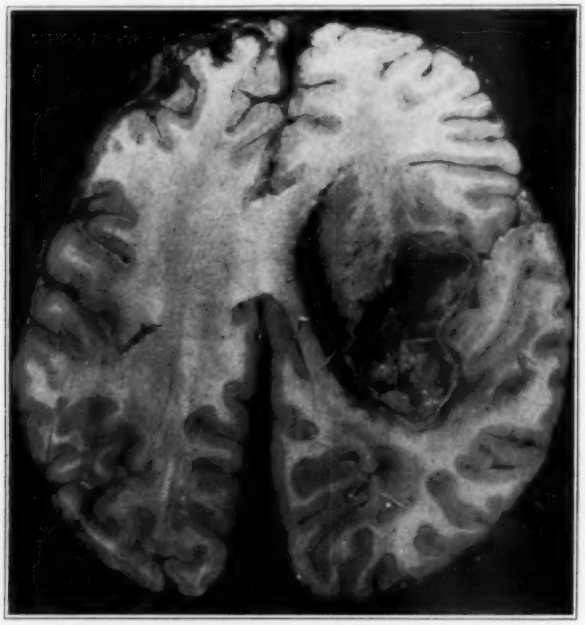

Fig. 28 (case 7).—Gross appearance and location of abscess. Note the well formed capsule.

newly formed blood vessels, formed a strong barrier between the abscess and adjacent brain tissue (fig. 29).

Comment.—Here again we see a rather typical series of events culminating in an abscess of the brain. Of interest is the formation of the capsule, which would indicate by its thickness the approximate length of time of its development. It may be said that the abscess preceded the terminal meningitis.

CASE 8.—*Patient had two admissions to the hospital. On first admission he displayed signs of meningitis and revealed purulent cerebrospinal fluid containing a poorly defined, gram-positive diplococcus. Antimeningococcus serum adminis-*

tered with apparently good results. On second admission, six months later, sudden relightening of an infection of the left ear with the development of symptoms of increased intracranial tension (but no papilledema or focalizing signs). Again purulent cerebrospinal fluid and an organism of a doubtful type were obtained. After a short period of improvement there followed an abrupt decline, with the development of localizing signs pointing to an expanding lesion in the left temporal lobe. No operation. Death. Necropsy.

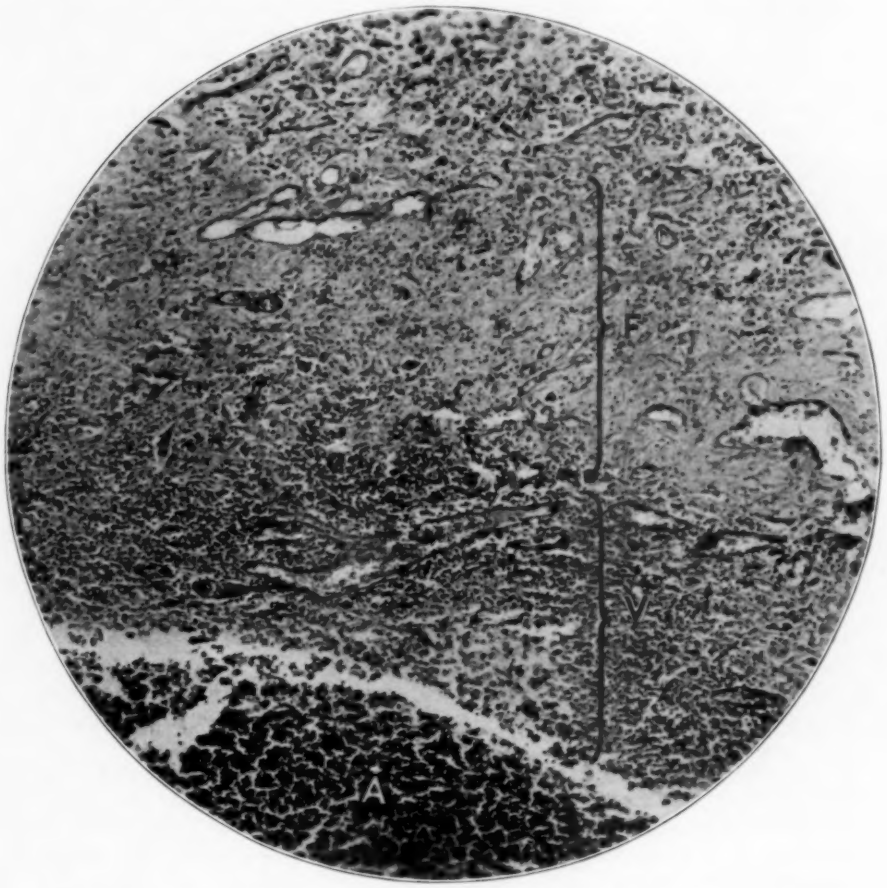

Fig. 29 (case 7).—Microscopic appearance of abscess capsule. *A*, abscess cavity; *V*, vascular zone; *F*, fibrous zone containing many vessels. Photomicrograph, × 75.

History.—In A. G., a boy, aged 6 years, one week prior to his first admission to the hospital there suddenly developed pain in the left ear, headache and a rise in temperature (from 102 to 103 F.). On the third day of the illness the right drum ruptured spontaneously and discharged pus. The pain disappeared and the temperature returned to normal. For two days the patient remained comfortable; then his temperature rose again to 102 F. and severe frontal headache reappeared.

Examination.—There was a mild rigidity of the neck, with bilateral Kernig and Babinski signs and hyperactive deep reflexes.

Course.—Meningeal irritation of an impending meningitis secondary to the otitic process was diagnosed. The right ear showed an inflammatory process but no evidence of mastoiditis. The left ear, because of the narrowing of the canal by an external otitis, could not be thoroughly examined. A lumbar puncture yielded cloudy cerebrospinal fluid, under normal pressure, containing 1,500 cells (75 per cent polymorphonuclear leukocytes). A smear of the fluid showed many intracellular and extracellular gram-negative diplococci, suggestive of meningococci, which did not grow in culture. The patient was given a puncture daily during the next five days and antimeningococcus serum. He seemed to respond favorably to the treatment as the temperature returned to normal; the headache disappeared and the meningeal signs lessened. The cell count in the cerebrospinal fluid also dropped; organisms could no longer be found in the smears, and the cultures of the cerebrospinal fluid remained sterile. On the eleventh day a serum rash developed, but otherwise the patient continued to improve. Cultures of the throat revealed staphylococci and streptococci. A roentgenogram of the mastoids showed underdeveloped, small and hazy cells, the left more so than the right. The patient was discharged on Dec. 31, 1928, twenty-five days after admission to the hospital, free from signs and symptoms.

Three months later he was seen at the follow-up clinic. The ears were normal. He felt well and remained apparently so for another two months, at the end of which time, i. e., six months after the onset of the illness, he was readmitted to the hospital because of a sudden recurrence of vomiting and an elevation of temperature. During the first two days in the hospital, the temperature remained high, but the vomiting stopped. On the third day the patient complained of pain in the left ear. A myringotomy was done and sanguinopurulent material was evacuated. The ear continued to drain for the next nine days. The temperature returned to normal for a few days, but rose again to 102 F.; the severe frontal headache and frequent vomiting returned. There appeared mild rigidity of the neck, a slight bilateral Kernig sign, hyperactive deep reflexes, a bilateral Babinski sign, and slight nystagmoid twitching on lateral fixation of the eyes. The pulse was slow. A lumbar puncture revealed slightly cloudy cerebrospinal fluid under moderately increased pressure, containing 400 cells (67 per cent polymorphonuclear leukocytes). A culture of the cerebrospinal fluid was negative, but smears showed many gram-positive cocci, chiefly extracellular. During the following four days lumbar punctures were performed daily, small quantities of cerebrospinal fluid removed and antimeningococcus serum administered. The cell count rose gradually. First, gram-positive organisms, then gram-negative, and finally no organisms at all were found in the smears of the cerebrospinal fluid. The cultures remained sterile.

Repeated neurologic examinations disclosed a bilateral Babinski sign, but no papilledema, meningeal signs, clouding of the intellect or localizing signs. The patient at first showed some improvement, so that on the fifth day a lumbar puncture yielded clear cerebrospinal fluid under normal pressure. Two days later, however, there appeared mild right-sided weakness of the face, an increased right ankle jerk and a questionable Babinski sign on the right side. The patient then began to lose ground, and there appeared bilateral papilledema and visual field disturbances in the character of a right homonymous hemianopia. Five days later the patient showed more pronounced papilledema and more definite hemianopia while the temperature remained low and the pulse slow. Two days later generalized convulsions developed, which were more marked on the right side. A lumbar

puncture revealed 5,000 cells but no organisms. The temperature rose, the patient's condition declined rapidly and a fatal issue ensued.

Necropsy.—The dura was under increased tension. The brain was voluminous. The vessels on the surface of the brain were injected. The pia-arachnoid was faintly cloudy. The left temporal lobe was bulging, soft and fluctuating. The leptomeninges at the temporal pole on the left side were adherent to the floor of the middle fossa. When this adhesion was broken, there was noted a circumscribed darkened erosion measuring about 1 cm. in diameter in the leptomeninges (fig. 30*a*). Aspiration of the large fluctuating mass in the temporal lobe yielded copious greenish purulent fluid. On section of the brain a large abscess cavity filled with purulent material was found in the left temporal lobe (fig. 30*b*). Section through the wall of an abscess showed advanced organizations typical of an abscess of old standing.

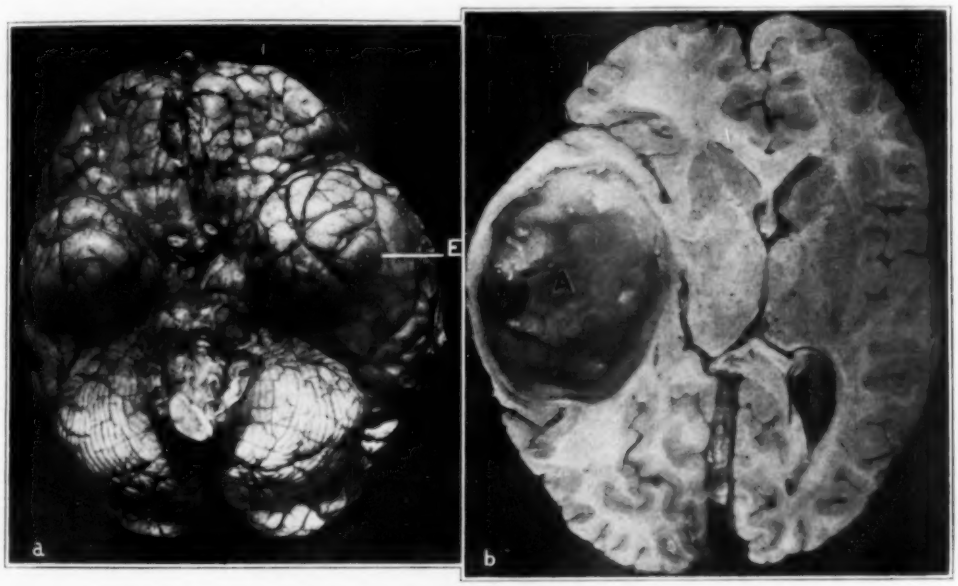

Fig. 30 (case 8).—In *a*, base of the brain showing bulging of the left temporal lobe and a circular area of denuded leptomeninges (*E*). In *b*, gross appearance and location of abscess. *A*, abscess.

Microscopic Anatomy: In this case there was found a very instructive histologic picture. The coats in the abscess were exceedingly well brought out, since they assumed their distinctive characters. The line of demarcation between brain tissue and abscess wall was well shown (fig. 31). The fibrous layer (fig. 32) was well demonstrated under higher magnification as consisting of young fibroblasts. The vascular layer (fig. 33) was rich and in large thin-walled vessels, surrounded by numerous lymphocytes.

Comment.—In this case there are several unusual features which demand interpretation. But as will be seen, no unqualified answer can be given to many of the questions that arise from the study of the clinical history. The most puzzling is the one concerning the organism

which provoked the early meningeal manifestations and very likely brought about the localized purulent encephalitis (abscess of the brain). At no time in the clinical course was the true character of the organism definitely recognized. It was reported during the early days of the patient's illness as a gram-negative diplococcus, and it was thought to be only suggestive of meningococcus, but what is rather important is that no growth could be obtained in cultures. This, however, does not

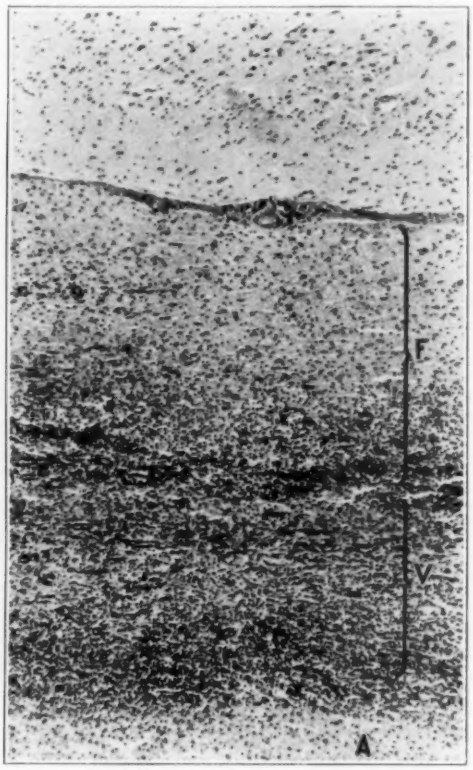

Fig. 31.—Histologic structure of the abscess wall. *A*, abscess cavity; *V*, vascular zone; *F*, fibrous zone. Photomicrograph, reduced from × 115. Note the line demarcating the abscess from intact brain tissue.

exclude the possibility that the causative organism was the meningococcus. The fact that the patient seemingly reacted well to antimeningococcus serum gave strong support to the diagnosis of epidemic cerebrospinal meningitis. At this time, of course, there was no suspicion of the formation of abscess of the brain since no localizing signs were manifest. The subsequent clinical course certainly revealed little to affirm the first diagnosis of meningococcus meningitis, since the

organism obtained early in the clinical course was gram-negative, but later in the course a gram-positive organism was obtained. The fact that still later no organism could be found in the cerebrospinal fluid would suggest the probability that there was a progressive degeneration of the organism responsible for the misleading staining properties with its final disappearance. It is is quite probable that the same condition occurred on the patient's first admission.

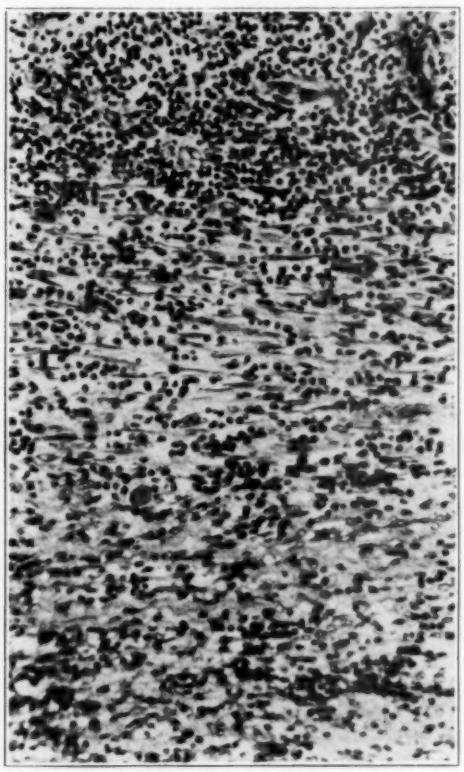

Fig. 32 (case 8).—The fibrous layer under higher magnification. Photomicrograph, reduced from × 300.

Should this explanation be allowed to stand, then, the course of events is better understood. The meningeal process abated a diffuse meningitis; at about the same time the infectious process extended into the depth of the contiguous brain tissue, leading to the formation of abscess. The thickness of the wall of the abscess indicated a fairly long-standing process, not unlikely dating back to the early clinical history. It is quite probable that it was initiated at the first admission and, having been walled off, remained dormant clinically until it began to produce signs of increased intracranial tension.

CASE 9.—*Bilateral otitis media following the grip. Mastoiditis. Double mastoidectomy and sinus exposure. Ligation of jugular vein. Exposure of the sigmoid sinus. Death. Necropsy.*

History.—B. R., a woman, aged 55, nine weeks before admission to the hospital, had the grip, with generalized pains and a rise in temperature for a period of two weeks. Following this, partial deafness in both ears with bilateral tinnitus developed. Shortly after, the patient began to have severe left-sided headache, with the aural disturbances becoming limited to the left side. Until three days before admission to the hospital the headache and aural disturbances had been almost constant

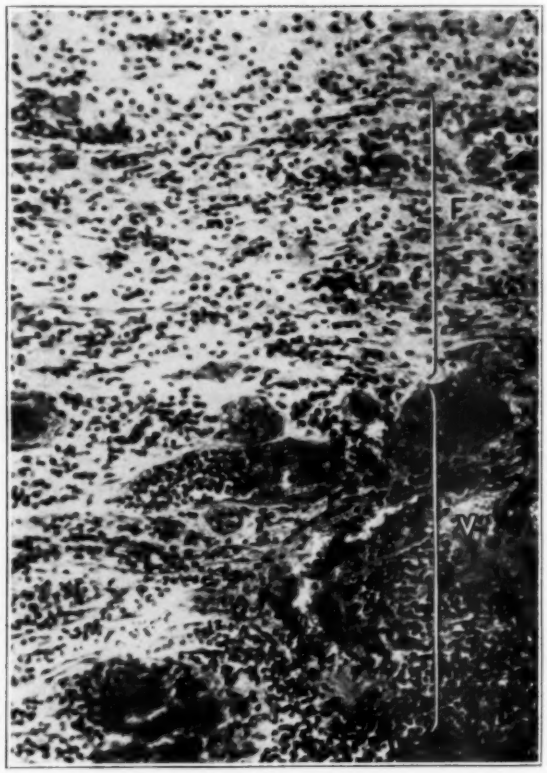

Fig. 33 (case 8).—The vascular layer (*V*) under higher magnification. *F*, fibrous zone. *V*, Photomicrograph, reduced from × 270.

and unchanging in severity. She ran a febrile course with a temperature ranging from 102 to 105 F. It was also noted that her gait was unsteady and that she was falling to the left.

Examination.—In the left ear the drum was thickened, the landmarks were not visible, there was no discharge and hearing was impaired. The right ear showed a bleb in the upper posterior quadrant of the drum, landmarks were absent, hearing was also impaired and there was tenderness over both mastoids.

Course.—In view of the history of an acute aural infection nine weeks previously, associated with headache and a rise in temperature, and a few focal signs,

the possibility of a latent mastoiditis with intracranial complications was considered. The white blood cell count was 10,600 with 74 per cent polymorphonuclears. The abdominal reflexes could not be elicited. There were no other neurologic signs. A lumbar puncture revealed clear cerebrospinal fluid under normal pressure and containing no cells. Six days after admission definite tenderness over the right mastoid developed. There was some sagging of the superior canal wall and a palpable, tender gland at the angle of the jaw on the right side. A blood culture revealed a pneumococcus, type III. The diagnosis of a right mastoiditis with sinus thrombosis was made, and the patient was operated on. A simple double mastoidectomy was done and the lateral sinuses were explored. The left mastoid showed disease and softening throughout. The sinus plate, however, was normal and intact. The left lateral sinus was then exposed and found normal. The right mastoid showed disease. The sinus wall was black and when opened accidentally did not seem to bleed freely. One week after the first operation the right jugular vein was ligated, with further exposure of the sinus from the lower end of the sigmoid to 2 inches (5 cm.) posterior to the knee. The sigmoid sinus was opened and a clot removed. The wound was reopened three days later and the bulb was drained. The patient became stuporous and died three days later (three weeks after admission).

Necropsy.—Gross Anatomy: Lying on the inner surface of the dura, along the posterior surface of the petrous portion of the temporal bone, there was a fairly firmly attached plastic exudate. A similar exudate was found in patches over the right cerebellar hemisphere and in the angle between the cerebellum and the right occipital lobe.

Microscopic Anatomy: A section of the cerebellar cortex adjacent to the localized meningeal process presented the appearance of early abscess formation characterized by thrombosis of vessels, extravasation of blood, necrotic changes in the tissue and a diffuse accumulation of polymorphonuclear leukocytes (fig. 34) and lymphocytes in the adjacent zone.

Comment.—This case serves as an excellent illustration of the initial stage in abscess formation. Here one sees several large extravasations surrounded by areas of perivascular infiltrations, which indicates an acute inflammatory process. It is quite probable that in every instance of abscess of the brain the initial stage is characterized by septic thrombosis of several large blood vessels followed by rupture of some of the radicals of these vessels with subsequent extravasation. The infectious material sets up an inflammatory reaction with synchronous central softening and peripheral granulation tissue formation.

CASE 10.—*Left otitis media, followed by intense headache, vomiting and unsteady gait. Cerebellar signs. Exploration disclosed a left cerebellar abscess. Death. Necropsy.*

History.—H. T., a boy, aged 11 years, had a cold and pain in the left ear six weeks prior to his admission to the hospital. Two weeks later the ear began to discharge profusely and continued to do so until three days before admission. For the past ten days he had had severe frontal headache. His gait became unsteady; he would stagger and fall to the right. He vomited once the day before admission. There was no elevation of temperature or vertigo. Five years previously the patient was treated for an infection of the frontal sinus, and at that time he suffered from severe headache.

Examination.—There was a large healed perforation in the right drum. The left ear had the appearance of a resolving infection of the middle ear, the light reflex was visible, the canal wall was normal and there was no mastoid tenderness or edema. The patient was able to hear a low whisper. There were no meningeal signs. There were a coarse nystagmus to the right, a fine nystagmus to the left and an occasional rotary nystagmus. All deep reflexes were hyperactive, the left

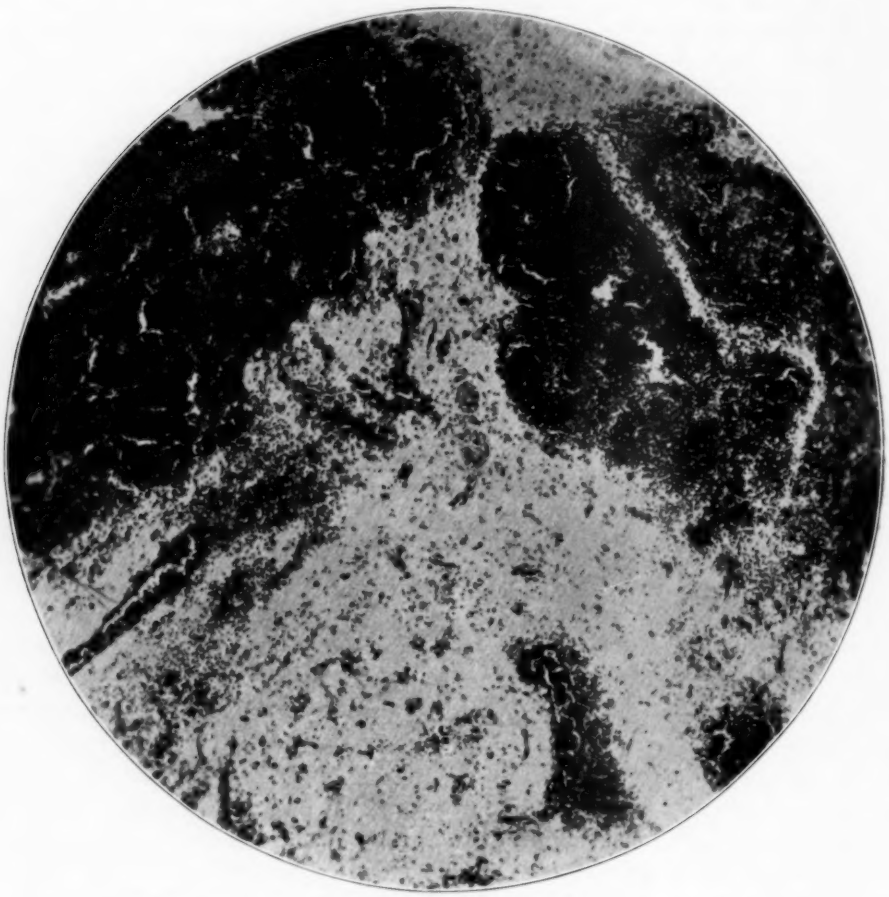

Fig. 34 (case 9).—Early abscess formation. Photomicrograph, × 75.

more than the right. There was no past pointing, but ataxia and adiadokokinesis in the left arm were present. The patient reeled to the right.

Course.—On the patient's second day in the hospital a vertical nystagmus developed in addition to the previously mentioned signs. This, coupled with the history of suppuration of the left ear, pointed to a lesion in the left cerebellar lobe, probably pressing on the brain stem. A lumbar puncture revealed cerebrospinal fluid under increased pressure containing 150 cells but no organisms. Two days after admission a suboccipital craniotomy was performed. At the depth of 2 cm.

pus was encountered in the left cerebellar lobe under great pressure. The situation of the lesion suggested that it was deep and close to the petrous pyramid. The abscess cavity was widely opened, drained and packed with iodoform gauze. The following day the frontal headache became more intense and the pulse slow (64 per minute). No other changes were noted in the patient's condition until the end of one week when he became irrational and showed a bilateral Kernig sign, a rigid neck and a rise in temperature to 104 F. He declined rapidly and died eleven days after admission.

Necropsy.—Gross Anatomy: There was an operative wound present in the left suboccipital region through which purulent material exuded, and when a wider exposure of this wound was made, necrotic brain tissue was exposed. The cerebellum disclosed a large abscess in the left lobe (fig. 35). It was surrounded by a wide zone of disorganized tissue. There was also a thickening of the meninges over the region of the abscess. Further study showed that the left cerebellar hemisphere was almost completely replaced by necrotic tissue which showed central destruction and was separated from the fourth ventricle by a narrow zone of

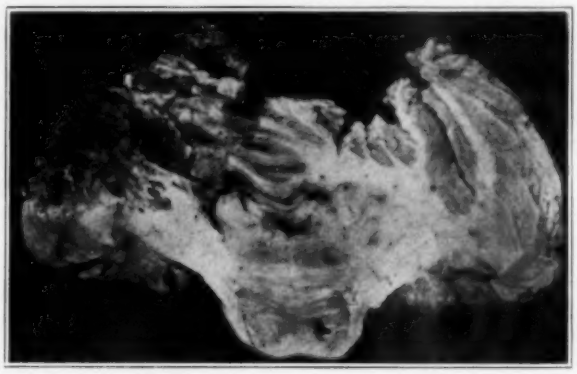

Fig. 35 (case 10).—Gross appearance and location of an early cerebellar abscess.

necrotic tissue. It appeared as though there had been some extension of the process into the floor of the fourth ventricle, as the latter was somewhat distorted.

Microscopic Anatomy: Sections showed a fairly diffuse leukocytic infiltration into the cerebellum with areas of recent hemorrhage (fig. 36). The surrounding zone was characterized by a moderate fibroblastic proliferation. Numerous blood vessels at a distance from the inflammatory process showed perivascular infiltration of small lymphocytes.

Comment.—Here again the localization of the lesions offered no difficulties. The unsuccessful surgical procedure does not provide any opening for criticism. It must be realized that in some of the abscesses, particularly in the very acute stage, any attempt, no matter how basically correct, is apt to result in failure. This is particularly true of cerebellar lesions, in which encapsulation is a much slower process.

CASE 11.—*Grip and pneumonia followed by repeated abscess formations in various parts of the body, including osteomyelitis of the sternum. Later, otitis media. Mastoiditis. Mastoidectomy. Signs of increased intracranial tension. No*

abscess. Irregular course with development of cerebellar signs and evidence of meningeal involvement. Cerebellar exploration. No findings. Radical mastoidectomy. No relief. Gradual decline. Death. Necropsy.

History.—M. M., a man, aged 23, had the grip in March, 1923, accompanied by pneumonia. In April, an abscess developed in the anterior wall of the chest which was incised and drained. It never healed properly. In May, a psoas abscess developed which was incised and drained. A culture of the pus revealed Staphylococcus aureus. On October 2 a blood culture was taken and revealed the same organism. The patient was discharged on October 25, with the wound slightly discharging. Within a few days he began to complain of tinnitus in the right ear and, one week later, of pain in the right ear and the right side of the head, which became steadily worse. Four days later, the ear began to discharge and the patient began to complain of nausea, vomiting and dizziness.

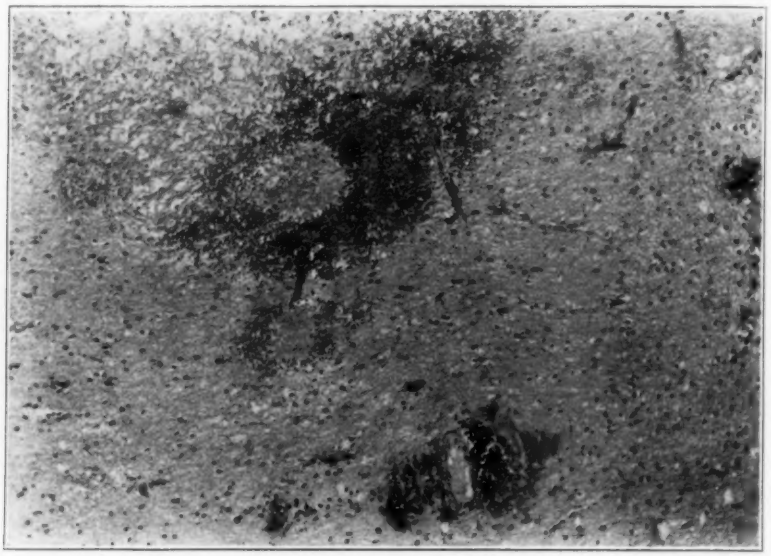

Fig. 36 (case 10).—Histologic appearance of the suppurative lesions. Photomicrograph, reduced from × 125.

Course.—He was readmitted on November 16. His temperature was 101.6 F., and he was drowsy and toxic. There were tenderness over the right mastoid, a discharge from the right ear, moderate nystagmus to the left, a right peripheral facial paresis and markedly impaired hearing in the right ear. Two days later right external rectus palsy developed and later hyperesthesia in the distribution of the right fifth nerve and complete deafness on the right side. A lumbar puncture revealed clear cerebrospinal fluid containing 27 lymphocytes. The white blood cell count was 11,000 with 80 per cent polymorphonuclear leukocytes. An exploratory mastoidectomy was done four days after admission. No pathologic alterations were found, no pus and no granulations. The middle fossa and the sinus were explored and found normal. Following the operation nystagmus developed in all directions. It was more marked to the left. The right pupil became larger than the left. Two days later there developed a more marked tendency to a bilateral Kernig sign, but no rigidity of the neck. Four days later the patient began to

show improvement of the right rectus palsy, but a low grade papilledema set in and with this the meningeal signs, such as the Kernig sign and rigidity of the neck, became more pronounced. The cerebrospinal fluid contained 675 cells with 90 per cent lymphocytes. With the advance of the meningeal signs, the local signs, such as the right rectus palsy, the right facial weakness and the sensory disturbances in the distribution of the fifth nerve, gradually cleared up. The meningeal signs persisted, but papilledema was slowly advancing. A week later a temporosphenoidal exploratory aspiration was done with negative results. The complete dysfunction of the right acoustic nerve persisted. Cultures of the blood and cerebrospinal fluid were now reported negative. Ten days later a cerebellar exploration was performed with negative results. The meningeal signs became more marked and the cerebrospinal fluid now showed 3,500 cells, with 90 per cent of polymorphonuclear leukocytes. Another month passed and a mastoidectomy was done. The general meningeal signs and symptoms persisted with occasional remissions and exacerbations for about one month. Then a marked improvement set in for a brief period, interrupted by the appearance of more definite cerebellar signs. These persisted, while the general condition improved greatly for about ten days. Then the patient began to decline again, with vomiting, increasing in frequency, apathy and severe headaches. Because of the persistence of the right cerebellar signs, right cerebellar aspiration was advised, but on May 12 there was a sudden rise in temperature, frank evidences of pneumonia appeared, pulmonary edema set in and the patient died, six months after admission to the hospital.

Necropsy.—A partially collapsed abscess in the right cerebellar hemisphere was found.

Microscopic Observations: Histologic examination of the abscess wall showed fairly advanced organization, with marked reduction in the abscess cavity (fig. 37).

Comment.—The early stages of the cranial nerve involvement, such as sensory disturbance in the distribution of the fifth nerve, total loss of hearing on the right side and marked nystagmus in all directions, should have pointed to localization in the posterior fossa rather than to abscess of the temporosphenoidal lobe. The moderate meningeal signs and the pleocytosis should have indicated the existence of a circumscribed meningeal process rather than a frank abscess. This will explain the failure to find an abscess in the temporosphenoidal lobe or in the cerebellar hemisphere. An abscess was later found in the left cerebellar hemisphere, but it must not be regarded as an extension of an earlier meningeal process, as testified by the early anatomic alteration in the wall of the abscess.

CASE 12.—*Acute mastoiditis. Mastoidectomy. Apparent recovery. Sudden onset of manifestations of increased intracranial tension. Signs and symptoms of cerebellar abscess. Exploration of cerebellum through trephine opening. Pus recovered. Temporary improvement. Second operation. No pus found. Decline. Death. Necropsy.*

History.—W. S., a boy, aged 6 years, had undergone a mastoidectomy seven weeks prior to admission to the hospital. During the first three weeks he convalesced uneventfully and appeared to have regained good physical condition. He remained well for another two weeks and then suddenly had a chill. The tempera-

ture rose and remained elevated during the following three days, ranging from 101 to 102 F. The patient complained of headache and was admitted to the hospital.

Examination.—The patient was drowsy, and the left ear drum flat and gray with the short process visible. The ear was dry, but a postauricular fistula was discharging slightly. Hearing was unimpaired. There was spontaneous past pointing to the left. Adiadokokinesis was marked. Dysmetria was present in the left upper extremity. The gait was guarded and there was a tendency to fall to the left. The deep reflexes were increased, the abdominal reflexes were diminished and a Babinski sign was elicited on the right side.

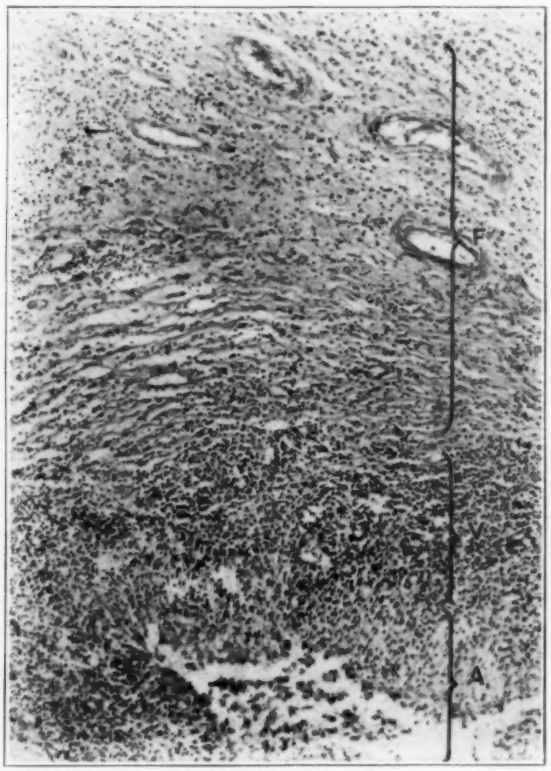

Fig. 37 (case 11).—Section of the abscess capsule, showing advanced organization. *A*, abscess cavity; *V*, vascular zone; *F*, fibrous zone. Photomicrograph, reduced from × 125.

Course.—The diagnosis was an expanding suppurating lesion in the left cerebellar hemisphere, and a search for a cerebellar abscess was made through a trephine opening in the mastoid region. An aspiration needle was inserted to a distance of 3 cm. from the surface, and about 4 cc. of pus obtained. A rubber tube was then introduced into the abscess cavity for drainage. On the day following the operation the child's condition was poor; he was listless and at times irritable. He vomited frequently. In addition to the neurologic signs previously recorded there was now weakness of the left arm and leg. During the following week there was noted, at first, moderate improvement in the child's condition, but it

was suddenly interrupted by the appearance of drowsiness, more pronounced rigidity of the neck and a bilateral Kernig sign. He vomited frequently and became more irritable and the headache became severe. The objective findings showed particularly no change except that the Babinski sign on the right side became more definite. The right optic disk began to display early papilledema. Paralysis of the left abducent nerve and facial weakness on the left side developed. The vomiting persisted. The papilledema of the right disk progressed, and there appeared an inexhaustible ankle clonus on the left side. A lumbar puncture revealed clear, colorless cerebrospinal fluid, containing 15 cells, but no organisms. In view of the persistence of increased intracranial tension, a second attempt to drain the cerebellar abscess was made. This time an incision was made through granulation tissue covering the original operative field. A needle was passed but no pus revealed. Following the operation the child became more irritable, the disk became more elevated and pain in the head became intense. To relieve the headache a ventricular puncture was done; 40 cc. of clear colorless cerebrospinal fluid was removed. The child reacted well to this procedure and seemed to improve, but three days later vomiting and headache recurred. Four days after the ventricular puncture the child was subjected to another exploratory craniotomy. The original trephine opening was enlarged and a portion of the herniating tissue resected. Again no abscess was found. The day following the operation ptosis of the left eyelid developed with marked internal strabismus. The temperature rose to 103 F., and the pulse became irregular. Signs of meningitis developed and a fatal issue followed two days later.

Necropsy.—Skull: There was a circular bony defect, 1 cm. in diameter, in the skull posteriorly. The dura was adherent to the skull near the margin of the defect. The left middle ear contained pus, which in smear showed many gram-positive "coccoid" organisms. Just behind the sigmoid sinus there was a small crescent-shaped defect in the thin plate of bone. This defect, 1 cm. in length, was separated from the mastoid sinus and the craniotomy wound. The right middle ear was normal.

Brain: The convolutions of the brain were flattened. The ventral surface of the tentorium cerebelli facing the right lobe of the cerebellum was coated with a thick layer of yellow pus which also covered the entire right cerebellum. The left cerebellar hemisphere presented on its surface diffuse areas covered with purulent exudate. There was an abscess cavity in the left cerebellar hemisphere (fig. 38) which extended from the surface of the cerebellum, where it was adherent to the cranial defect, deep into the central part of the lobe. The left cerebellar lobe was distinctly larger than the right.

Microscopic Examination.—Brain: Histologic examination showed evidence of a purulent inflammatory process in the left cerebellar lobe. The process was characterized by the presence of numerous polymorphonuclear and mononuclear leukocytes and by marked proliferation of fibrous tissues. The blood vessels throughout the region showed marked infiltration of the adventitial coats and the perivascular spaces with lymphocytes. The meninges likewise showed evidence of a chronic inflammatory process. Other sections showed advanced organization of an abscess wall with a thick fibrous capsule, indicating a fairly advanced healing process in the capsule (fig. 39).

Comment.—In this instance the diagnostic features require no discussion. A typical clinical picture of cerebellar abscess was presented and recognized. The surgical steps, however, judging from the results obtained, cannot be regarded as best adapted to a lesion of this type.

The attempt to reach an abscess through a trephine opening is certainly not a procedure of great precision. Blind searching and inadequate protection against spread of the infection by the aspiration needle, followed by unavoidable ignorance as to what was accomplished, are its only rewards. Realizing that in the present circumstances there is little more than a slender chance for recovery from an abscess of the brain, what is there to lose if a more radical approach is to be followed?

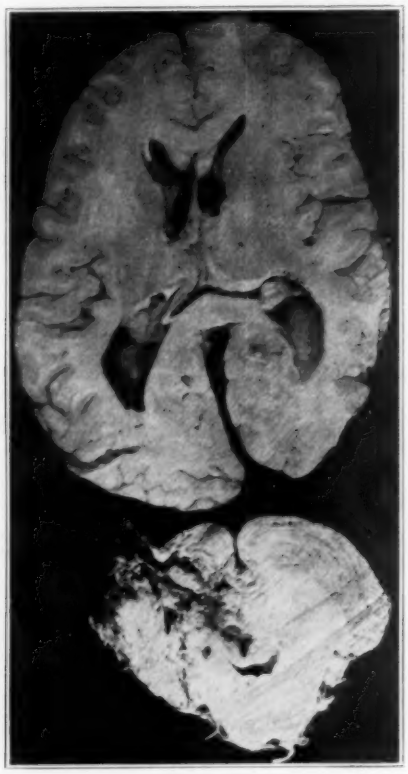

Fig. 38 (case 12).—Photographs illustrating the gross appearance of an abscess in the cerebellar hemisphere and fairly advanced internal hydrocephalus.

Better exposure, more complete exploration and more adequate treatment, even to the extent of a lobectomy, when carried out carefully and cautiously, cannot add to the existing hazards. Radical as it may be the procedure adds little to the factors determining a fatal issue, but it may open greater possibilities for a more rational treatment of cerebellar abscess.

CASE 13.—*Mastoiditis on the right side following scarlet fever. Sudden onset of intense headache, vomiting and a moderate elevation of temperature: Cere-*

bellar signs and manifestations of meningeal irritation. Craniotomy. Right cerebellar abscess found. Temporary improvement. Recrudescence. Revision of operation. Abscess found in field of previous operation. Rapid decline. Death. Necropsy.

History.—In B. R., a boy, aged 8 years, mastoiditis on the right side developed following scarlet fever. Three weeks prior to admission to the hospital, mastoidectomy was performed. It was followed by an apparently uneventful convalescence, but two weeks following the operation the patient suddenly began to complain of frequent attacks of frontal headache, often precipitated and intensified by sudden movements of the head. This was soon followed by frequent vomiting. The headaches became progressively more intense and more constant. The temperature rose to 101 F.

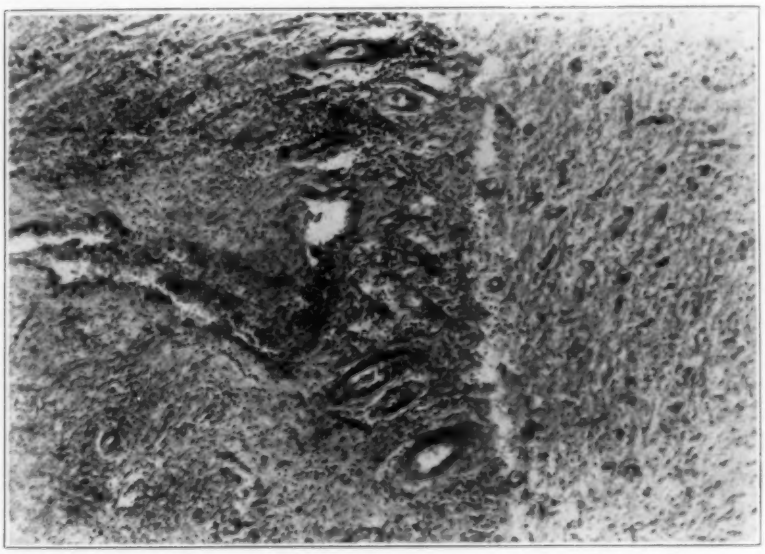

Fig. 39 (case 12).—Histologic appearance of the outer zone of the abscess wall, showing advanced organization. Photomicrograph, reduced from × 125.

Examination.—The patient was apathetic and apparently suffering from headache and dizziness. The fundi showed fulness of the veins and moderate blurring of the nasal margin of the left disk. There was a horizontal nystagmus, more marked to the right. There were dysmetria and ataxia on the right side. The abdominal reflexes were diminished on both sides. The gait was unsteady with a tendency to fall to the left. There was a healing right mastoid wound with a fistula at its lower margin. The drum was flat and there was no discharge. The hearing was slightly impaired on the right. A lumbar puncture yielded clear cerebrospinal fluid under increased pressure, containing 64 cells.

Course.—The patient's headache increased in intensity. Some retraction of the head, weakness of the right side of body and an increase in the swelling of the disks appeared. The diagnosis of an abscess in the right cerebellar lobe was made. Craniotomy was performed and a right cerebellar abscess was found and drained. Following the operation the patient began to improve. Convalescence was rapid

and he was soon allowed out of bed. However, his gait remained awkward and he still had some nystagmus on lateral fixation. The right knee jerk and ankle jerk were absent. The ataxia in both upper extremities was present, though less marked. The patient was allowed to go home. His condition remained stationary for about seven weeks, at the end of which period he began to vomit and to complain of headache and dizziness. He became drowsy.

Examination on Second Admission.—There was increased swelling of the disks: 2 diopters in the right eye and 3 diopters in the left. There was nystagmus in both directions, more marked to the right upper extremity. Another operation was performed. The abscess was found and drained. For one week the patient seemed to improve, but began to lose ground again, and signs of a recurrent suppurative process in the brain reappeared. Eighteen days later the patient was again operated on, and an abscess was again found in the region of the previous operation. Following the last operation, the temperature rose and the patient died twelve hours later.

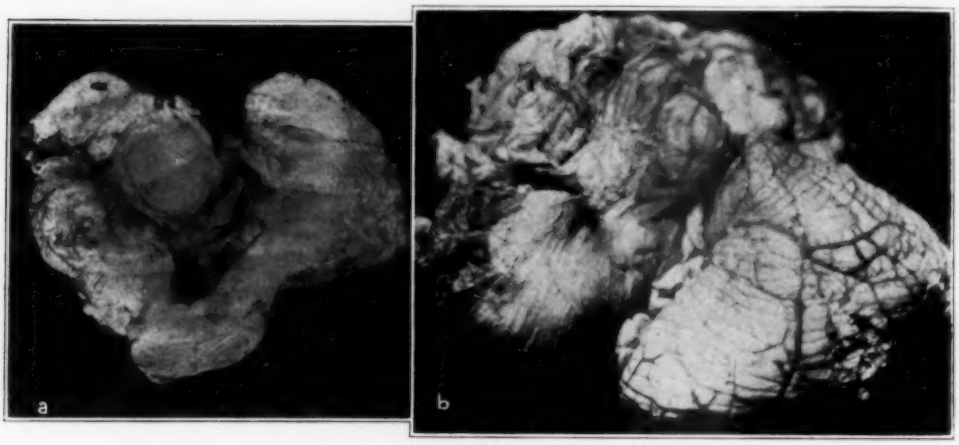

Fig. 40 (case 13).—In *a*, gross appearance and location of abscess. In *b*, appearance of the thickened leptomeninges over the abscess.

Necropsy.—Gross Anatomy: The brain was voluminous and showed evidence of intracranial tension. The meninges showed moderate dulling in places, but no frank inflammatory changes except for the right cerebellar hemisphere over its posterior lobe, where the meninges were scarified and adherent to equally indurated cerebellar tissue (fig. 40 *b*). On incision a well encapsulated abscess was found (fig. 40 *a*) in the right cerebellar hemisphere. It measured 2 cm. in the anteroposterior diameter. There was another area of disorganization lateral to the abscess formation. Posterior to the abscess the cerebellar cortex was replaced by a narrow band of scar tissue.

Microscopic Anatomy: The wall of the abscess displayed far advanced organization (fig. 41) with dense collagenous fibers, and enveloped an aggregation of leukocytic elements which were undergoing disintegration. The meninges covering the tissue adjacent to the abscess showed chronic inflammatory alterations.

Comment.—This case is instructive mainly for its anatomic alterations which developed following the operative procedure. It shows

definitely that the healing process was in progress and that the granulating tissue resulting from it contributed its share to the causation of the increased intracranial tension. It opens the question of whether the surgical procedure during the last craniotomy was the one best adapted to the case. Realizing that a healing and walling-off process had gone on for a long period of time, would it not have been more proper to attempt a lobectomy in the hope of accomplishing something that would benefit the patient rather than to attempt to drain the thickly encapsulated abscess, which is little more than a gesture? From the diagnostic point of view, there is little to add.

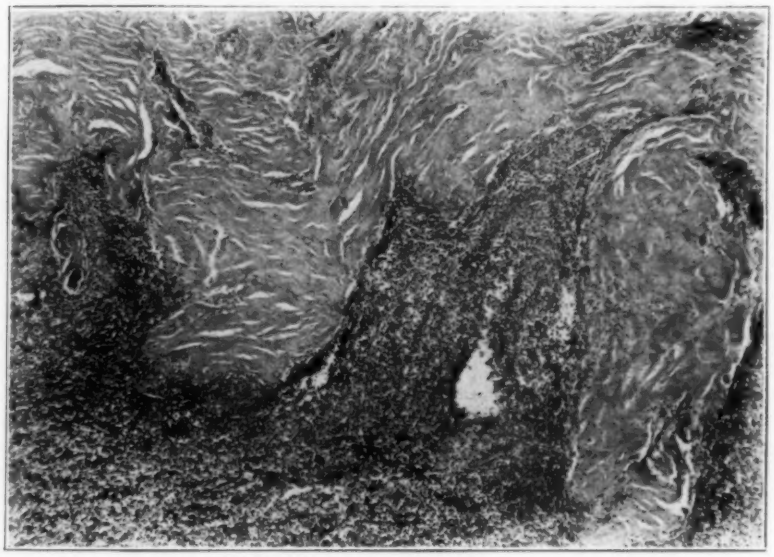

Fig. 41 (case 13).—Dense fibrous capsule of abscess. Photomicrograph, reduced from × 65.

CASE 14.—*Relightening of a chronic purulent otitis media, followed by a lateral sinus thrombosis. Jugular ligated and lateral sinus opened. Symptoms of increased intracranial pressure and signs of circumscribed expanding lesions in the temporal lobe. Several unsuccessful attempts to find and drain the abscess. Meningitis. Death. Necropsy.*

History.—In C. K., a youth, aged 16, two months before admission to the hospital, a mild sore throat and discharging left ear developed. The latter was a relightening process of chronic infection of the ear which had recurred intermittently in the course of the previous year. At the end of two weeks the patient's temperature acquired a septic character, with daily fluctuations ranging between 99.6 and 105 F., and a mastoidectomy was performed. As the septic character of the temperature continued the lateral sinus was opened and the jugular vein ligated. Streptococcus nonhemolyticus was recovered from the clot in the sinus. Following the second operation the temperature was lowered somewhat, ranging

between 97 and 102 F. Four days after the jugular ligation headache developed, and hemorrhages were noted in the left fundus. During the next two weeks the disks displayed advancing bilateral papilledema. The right knee jerk and ankle jerk became more active than the left and a right ankle clonus was observed. This was soon followed by jacksonian convulsive seizures on the right side, by transient attacks of aphasia and by a right facial paralysis. The patient was then brought to the hospital.

Examination.—The patient was drowsy. His temperature was 102 F., and his pulse rate 108. The left ear drum was flat and had a perforation in the posterior angle. The postauricular wound was open and discharging. The pupils were dilated, but reacted well to light and in accommodation. There was early bilateral papilledema. There were temporal anomia, slight astereognosis of the right hand, right-sided hyperreflexia with a Babinski sign on the right side and bilateral exhaustible ankle clonus.

Course.—The aural findings, the headache, the neurologic signs and symptoms, such as right-sided hyperreflexia, right facial weakness, right jacksonian attacks and the temporal anomia, suggested the diagnosis of an abscess in the left temporal lobe. An exploratory craniotomy was done through the mastoid defect. No pus was found. Following the operation the patient showed moderate improvement; he became more alert, the anomia disappeared and the neurologic signs became apparently less marked. The improvement continued for about one week, at the end of which time he became drowsy again and intense headache reappeared. The paralysis of the face became more pronounced and there appeared slight deviation of the tongue to the right. This was accompanied by a definite advance in the blurring of the left disk. Two days later the patient had a chill with a rise in temperature to 106 F. A blood culture taken at this time was positive for a gram-positive coccus. Two days later there developed paresis of the right upper extremity, right homonymous hemianopia and complete motor aphasia. The diagnosis of an abscess of the left temporal lobe was again made and the temporal lobe was explored through a trephine opening. A thin brownish fluid was obtained through the aspiration needle and a tube was inserted for drainage. Again there was gradual improvement, at first, with a return of power in the right upper extremity, partial return of speech and the disappearance of the hemianopia. But this improvement was soon arrested by the appearance of signs of a progressive decline in the patient's condition. The disturbance in speech became more pronounced, the headache more intense and the papilledema more advanced. A lumbar puncture revealed clear cerebrospinal fluid under increased pressure, containing 220 cells, mostly polymorphonuclear leukocytes. Another exploration through a small trephine opening in search of a circumscribed abscess failed to disclose such a process and was followed by the development of signs of diffuse meningitis. The patient died five weeks after admission.

Necropsy.—In the process of the opening of the skull, pus emerged through the trephine opening. The dura around the trephine opening was covered by purulent exudate. Some of the exudate was also found posteriorly over the temporal and occipital lobes. In the region of the left frontal lobe the dura was bulging, but on opening the dura, this area of the brain was found to be depressed, causing a concavity of about 4 cm. in the longitudinal and 6 cm. in the horizontal diameter; the space formed by the depression was filled with a large amount of pus (fig. 42 *a*). The mesial wall of this cavity was lined by moderately thickened pia and some disrupted cerebral cortex (fig. 42 *b*). Its other boundaries were formed by adhesions of the dura to the underlying brain substance. A thin plastic exudate

was found covering the meninges on the left side of the base of the brain, the dorsolateral surface of the right cerebral hemisphere. The dura and the leptomeninges over the inferior surface of the left temporal lobe presented a thick matted character, being adherent to the underlying roof of the temporal bone. At this point (the tegmen tympani) there was definite softening of the bony structure. The left lateral sinus was completely occluded by a thrombus.

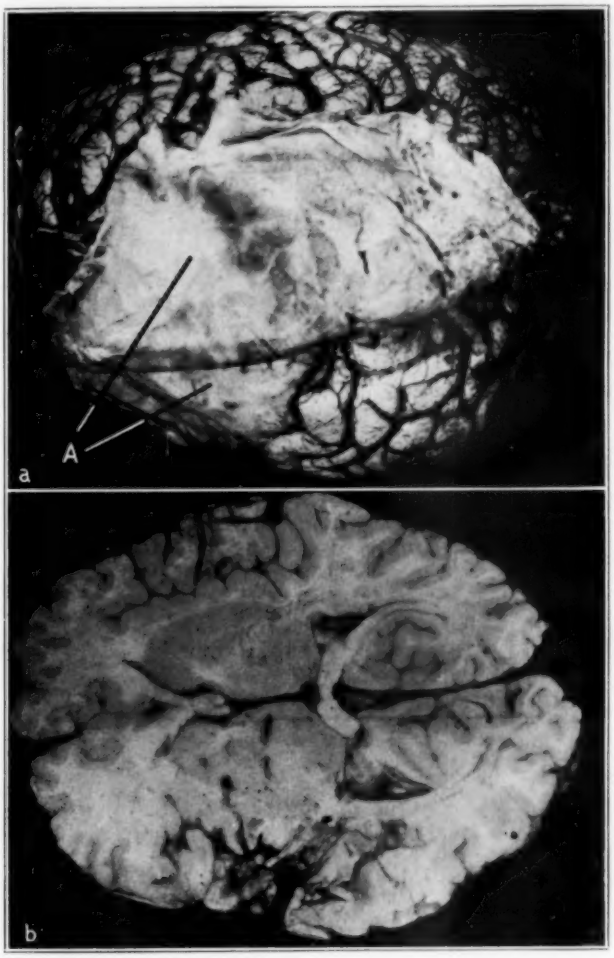

Fig. 42 (case 14).—In *a*, the gross appearance of the subdural abscess (*A*). In *b*, the gross appearance of the mesial boundary of the subdural abscess, showing disintegration of the cortex.

Microscopic Anatomy: The dura underlying the abscess displayed moderate inflammatory changes (fig. 43 *a*). The pia-arachnoid showed fairly advanced productive meningitis (fig. 43 *b*). In the depth of the disintegrated cerebral cortex extension of the meninges showed an outspoken process of organization (fig. 44 *a* and *b*).

Comment.—This case serves as a good illustration of what may be recognized as an intracranial abscess of the type termed by Eagleton [5] intrameningeal. Clinically it presents almost all of the features of an intracerebral circumscribed suppurative process. This case points again accusingly at the antiquated methods of approach in conditions of localized intracranial suppurative lesions. A wide exposure and a careful examination of the surface of the brain should certainly be the first

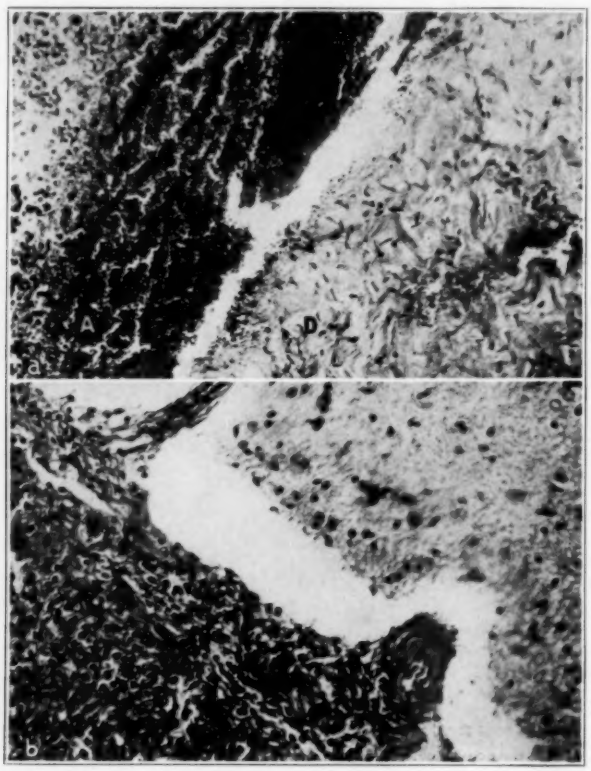

Fig. 43 (case 14).—In *a*, the dura (*D*) covering the abscess. Photomicrograph, reduced from × 250. In *b*, the pia-arachnoid overlying the abscess. Photomicrograph, reduced from × 340.

step taken before the damaging and threatening invasion of the brain by the exploring needle is attempted. It is logical to assume that a more satisfactory result could have been attained in this instance had the suggested method of investigation been employed.

CASE 15.—*Relightening of an old purulent otitis media. Mastoiditis. Radical mastoidectomy. No relief. Ligation of the jugular and opening of the lateral sinus. Signs of a focal suppurative lesion in the brain. Decline. Death. Necropsy.*

History.—S. R., a man, aged 60, had had a discharging right ear since the age of 4 years. Three weeks before his admission to the hospital the ear suddenly and spontaneously ceased to discharge. Simultaneously he began to experience severe pain in the right side of the head. Two weeks later, he suddenly became acutely ill with a chill, a rise in temperature and an increase in the intensity of the headache. Three days later he had another chill, vomited twice and became drowsy.

Examination.—The patient was drowsy. There were moderate ptosis of the left upper eyelid, slight rigidity of the neck and a bilateral Kernig sign. All the deep

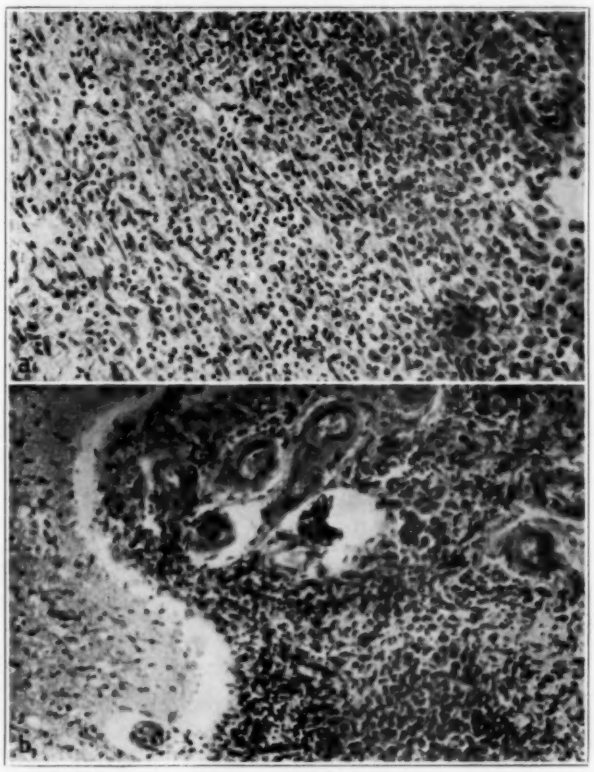

Fig. 44 (case 14).—In *a*, pia-arachnoid in the depths of disintegrating cerebral cortex. Photomicrograph, reduced from × 290. In *b*, fibroblasts in the membrane walling off the abscess. Photomicrograph, reduced from × 80.

reflexes were depressed and the left abdominal reflexes were absent. The right ear revealed a boggy canal and a foul smelling pulsating discharge in the region of the attic. The right mastoid was tender at the tip and there was loss of hearing on the right side. The left ear was normal. Caloric tests of the right ear revealed turbid cerebrospinal fluid under normal pressure. It contained 6,900 cells, mostly polymorphonuclear leukocytes and a few gram-positive organisms.

Course.—In view of the negative cultures of the cerebrospinal fluid and because of the persistence of headache, tenderness over the mastoid and the inactive laby-

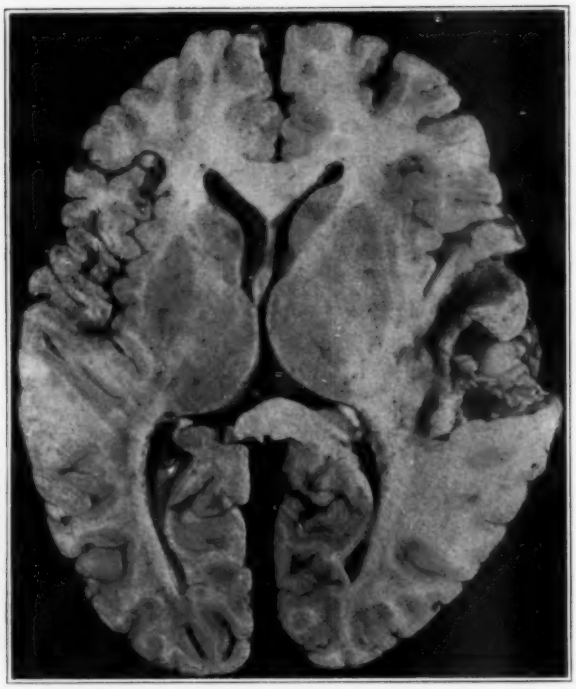

Fig. 45 (case 15).—Gross appearance of the areas of disintegration, simulating abscess of the brain.

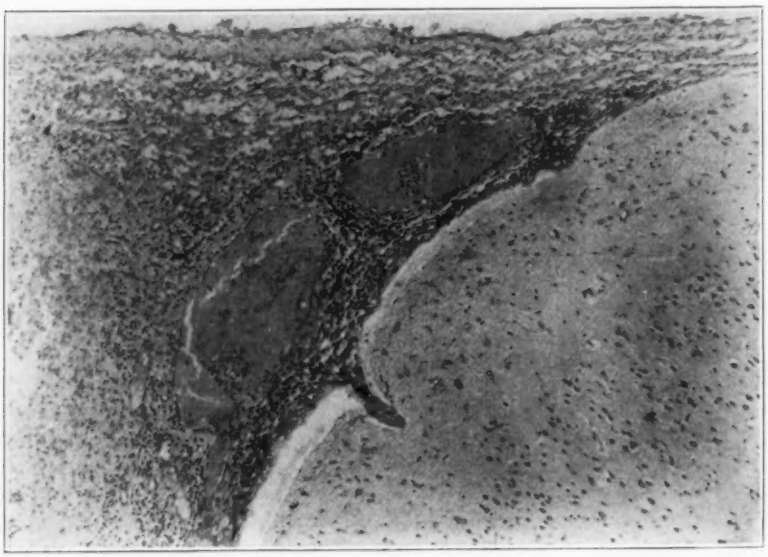

Fig. 46.—Purulent meningitis adjacent to the area of disintegration. Photomicrograph, reduced from × 115.

rinth, it was found advisable to explore the mastoid and labyrinth. The mastoid cavity was found to be small and sclerosed. Foul purulent material was found in the antrum. The entire plate of the middle fossa was absent; the dura was covered with a greenish exudate, and pus was seen to escape through a minute opening in the dura. The opening was enlarged and a rubber tube was inserted. On the following day the patient seemed to improve, he was bright and the headache was

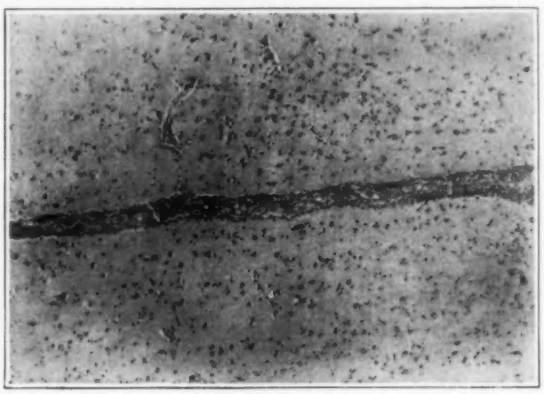

Fig. 47.—Extension of inflamed meninges into the involved brain substance. Photomicrograph, reduced from × 115.

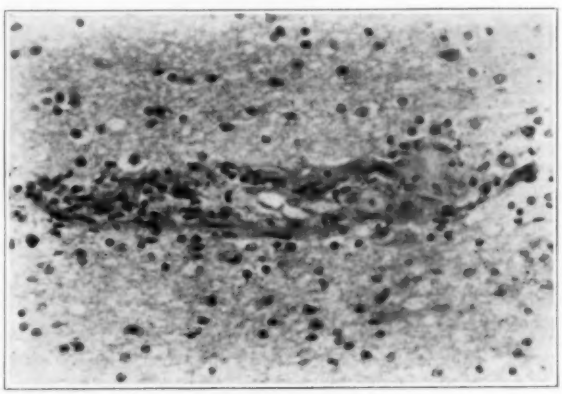

Fig. 48.—Center of disintegrating tissue, showing glial proliferation and leukocytic infiltration. Photomicrograph, reduced from × 400.

less severe. This improvement, however, was only temporary, as it was soon interrupted by a sudden chill and rise in temperature (104 F.). This, in the absence of neurologic manifestations, suggested the probable existence of sinus thrombosis. The jugular vein was ligated and the lateral sinus was opened. Following the second surgical procedure, the patient's condition continued to decline. There developed bilateral drooping of the eyelids and left hemiparesis with a left-sided pyramidal tract sign, including the Babinski sign. The fundi remained normal.

A lumbar puncture again revealed cloudy cerebrospinal fluid containing 600 cells, all of which were lymphocytes. Pulmonary signs developed which were interpreted as due to a metastatic abscess in the lung. Death occurred five weeks after admission.

Necropsy.—On removal of the calvarium the dura was found adherent to the inner surface of the petrous portion of the temporal bone and to the edges of the surgical defect. The leptomeninges showed only a small amount of purulent exudate, at the right of the sylvian fissure. The right temporal lobe was soft and displayed a ragged opening extending into the brain substance in the area of the second temporal convolution. The cerebellum at the lateral margin of the right lobe showed some disorganization and was covered by a purulent exudate. The right lateral sinus from the knee to the torcular herophili was occluded by a large adherent clot. On section of the brain an abscess (?) (fig. 45), measuring about 3 cm. in diameter, was found in the right temporal lobe.

Microscopic Anatomy: The meninges over the abscess cavity showed marked infiltration with polymorphonuclear leukocytes (fig. 46). What appeared on gross examination as an abscess cavity proved to be an area of disintegration of the cerebral cortex, with mild inflammatory changes. The latter could be traced to extensions of the meninges (fig. 47) into the depth of the brain substance. In the center of the area of disorganization mild glial proliferation and leukocytic infiltration was noted (fig. 48) as the only indication of an inflammatory process.

Comment.—This case is not unlike the previous one, except that the subdural process was associated with a large intracranial suppurative process. Here, very likely, the late manifestations of a localizing focus were due to the fact that the suppuration of the brain set in late in the course of the disease.

SUMMARY AND COMMENT

The clinical points of interest in the fifteen cases of abscess of the brain herein described have already been discussed and commented on in detail as each individual case was presented. Little is to be gained from recapitulating the diagnostic difficulties and the therapeutic obstacles. They do not lend themselves to generalization or additional interpretations except for the fact that a survey of the clinical histories reveals the encouraging observation that the diagnosis of the character and seat of the lesion was in the majority of instances correctly made, but also the sad realization that therapeutic measures were uniformly unsuccessful. It seems to us that satisfactory progress in the surgical treatment of abscess of the brain is more likely to be made if attention is directed to a better understanding of the progressive cellular alterations which take place in the evolution of the abscess capsule. They constitute an important phase in which mesodermal elements are mobilized as defensive forces for the isolation and limitation of a suppurative focus in the brain, a process which should be made to serve the therapeutic needs in the cure of abscess of the brain. Even in the very early stage of focal suppuration in the brain, the beginning of capsule forma-

tion may be discerned. It is characterized by proliferation of blood vessels and migration of lymphocytic elements into the zones surrounding the suppurative focus. This, as already noted, is shortly followed by the metamorphosis of lymphocytes into fibroblasts and by progressive organization of the changing extravasated elements, with the result that a distinct protective wall is formed, separating the intact brain from the abscess.

If the clinical conditions are satisfactory and do not interrupt further development of the capsule, the latter continues to grow in thickness, acquires a dense fibrous character and provides the tissue adjacent to the abscess with excellent protection against the spread of the purulent process. This is well demonstrated by the absence of frankly suppurative lesions in the vicinity of the abscess. The occasional lymphocytic infiltrations and the moderate gliosis found in these areas need not be considered as an extension of the infection, for they are merely additional reactive processes which participate in the construction of the limiting membrane.

It would seem that *time* is the element most essential for the proper development of the capsule. Thus, when by virtue of the location in a silent area of the brain the abscess does not disrupt normal function of the brain or provoke, by obstructing the flow of cerebrospinal fluid, signs and symptoms of rapidly advancing intracranial tension, and hence does not press for immediate surgical intervention, the conditions are most favorable for an uninterrupted growth of the capsule. This is particularly true of abscesses of the frontal lobe. They serve as good illustrations of what may occur if an abscess which, in the early stages of its formation, does not provoke grave cerebral signs and symptoms is left undisturbed. If left alone, its encapsulation goes on uninterruptedly with progressive reduction in size and occasional ultimate healing of the abscess.

The question arises, however, as to what extent one can rely on the process of encapsulation in the treatment of every variety of abscess of the brain. It is partially answered by Dandy who took into account the inherent potencies of the capsule to limit and heal a cerebral suppurative process. His method provides *time*, during which normal function of the brain may be preserved by reducing intracranial tension, avoiding the spread of the suppurative process often provoked by the antiquated technic and permits of further display of the healing properties of the capsule. It is not impossible that in Dowman's [6] method time is also gained. The importance of the time element becomes

6. Dowman, Charles E.: The Treatment of Brain Abscess by the Induction of Protective Adhesions Between the Brain Cortex and the Dura Before Establishment of Drainage, Arch. Surg. **6**:747 (May) 1923.

obvious when it is realized that the disturbance of the cerebral function, in the majority of instances, is due, not to the suppurative nature of the lesion, but to its tumor-like expanding character. It is the increasing intracranial tension which calls for urgent relief and often forces premature action and not the need for limitation of the suppuration. The latter is well taken care of by the capsule of the abscess.

The method of repeated tapping of abscess of the brain suggested by Dandy appears theoretically to meet this demand. It aims to remove one of the most disturbing factors in abscess of the brain, the cause of increasing intracranial tension, and in this manner to provide the *time element* necessary for a satisfactory evolution of the capsule. Though the purely technical side of this method is open to revision and may not be employed with satisfactory results in cerebellar abscesses, it nevertheless provides for the supertentorial lesion a new surgical approach which has a firm anatomic basis and, when improved, is likely to be productive of far-reaching results. It may, at the proper time, be followed by more radical procedures, such as the production of aseptic meningeal adhesions preparatory to more complete drainage or, as in cerebellar abscesses, to a lobectomy. But, whatever method is employed, in abscess of the brain the foremost surgical indication is first to establish and maintain normal intracranial tension until the encapsulation is sufficiently advanced to permit of a more adequate and, if need be, more radical surgical procedure.

RELATION OF VITAMINS A, D, B AND G TO OTOLARYNGOLOGY

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This investigation has been undertaken to determine the effect of a deficiency in vitamins A, D, B and G on the ear, nose and throat. The methods used in the study have been a comprehensive but incomplete review of the literature and animal experimentation with clinical application of the facts learned therefrom and from my practice. The literature on vitamins [1] is enormous, and contains fascinating by-paths in many subjects. All of these that do not bear directly on otolaryngology have been avoided. The practical value of animal experimentation and the clinical application of the findings are mutually dependent.

REVIEW OF LITERATURE

Vitamin A.—A diet deficient in vitamin A produces a metaplasia [2] in the epithelium of the nasal mucosa. This change is from the cylindric to the stratified squamous cells. It begins in a group of cells deep in the epithelium, which by enlarging and fusing with other similar foci extend toward the surface. Here they undermine the normal cylindric cells that are replaced by stratified squamous epithelia of the keratinizing type. At this time, or shortly thereafter, appear the first evidences of infection. The resulting pathologic condition is primarily from a nutritional disturbance, but subsequently is modified by inflammation. This nasal metaplasia is a prototype of that occurring in the epithelium at other parts of the respiratory tract, of the eyes, the salivary glands and

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From the Houston Eye, Ear and Throat Hospital and Rice Institute.

Presented at the annual meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, N. J., May 25, 1932.

1. McCollum, E. C., and Simmonds, N.: The Newer Knowledge of Nutrition, ed. 4, New York, The Macmillan Company, 1929.

2. Wohlbach, S. B., and Howe, P. R.: Tissue Changes Following Deprivation of Fat Soluble A Vitamin, J. Exper. Med. **42**:753 (Dec.) 1925. Tyson, M. D., and Smith, A. H.: Tissue Changes Associated with Vitamin-A Deficiency in the Rat, Am. J. Path. **5**:57 (Jan.) 1929. Goldblatt, H., and Benischek, M.: Vitamin-A Deficiency and Metaplasia, J. Exper. Med. **45**:699 (Nov.) 1927. Manville, T. A.: Pathologic Changes Occurring in White Rats Raised on Diets Deficient in Vitamin A, Arch. Int. Med. **35**:549 (May) 1925.

the genito-urinary system in several laboratory species [3] and in man.[4] Consequently, it can now be said that vitamin A is a biologic necessity to certain epithelia.

These changes are produced by diets so deficient in this vitamin that death occurs in the rat within from two to six months. Another variation [5] of tissue has been reported in which the amount of vitamin A in the diet is ample to support life, but is insufficient to maintain a normal epithelium. The latter type is a proliferation of the epithelium and is noted as a polyposis of the trachea (fig. 1). The polypi so closely resemble hypertrophy of the tonsil that an inadequacy of vitamin A is suggested as a cause of the latter condition. Metaplasia and hyperplasia of the epithelium are the only specific reactions of the tissue to insufficiency of vitamin A and inflammation is a concomitant finding.

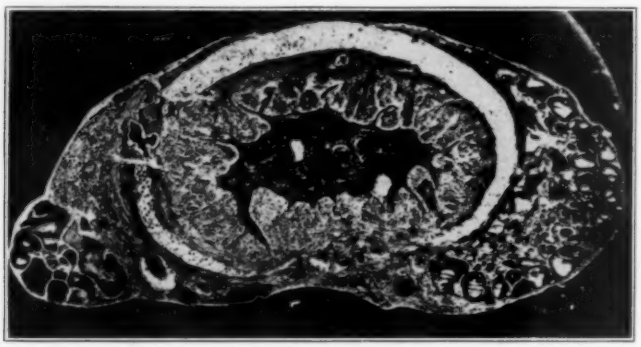

Fig. 1.—Hyperplasia of tracheal mucosa in a rat on a diet deficient in vitamin A for a year. The polyposis resembles hypertrophy of the adenoids. (After McCarrison.[5])

Xerophthalmia in animals and in man is the only characteristic disease of A avitaminosis. However, several conditions of the ear, nose and throat having ordinarily another etiology occur regularly as a consequence of the deficient diet. These conditions are suppurative sinusitis, suppurative otitis media and abscess of the salivary glands. The symptoms commonly found are nasal discharge, dysphagia, anosmia and impaired hearing. The dysphagia follows the abscesses of the submaxillary and lingual glands and is of prime importance in the final nutritional failure. Anosmia is the effect of metaplasia of the olfactory

3. Wohlbach, S. B., and Howe, P. R.: Vitamin-A Deficiency in Guinea-Pig, Arch. Path. & Lab. Med. **5**:239 (Feb.) 1928.

4. Wilson, J. R., and Dubois, R. O.: Report of a Fatal Case of Keratomalacia in an Infant, with Postmortem Examination, Am. J. Dis. Child. **36**:431 (Nov.) 1923.

5. McCarrison, R.: Effect of Insufficiency of Vitamin A on Tracheal Mucous Membrane and Thyroid Glands, Indian J. M. Research **18**:693 (Jan.) 1930.

epithelium. The aphonia is caused by the extreme dryness of the larynx, together with the mouth and pharynx. The sinusitis and otitis media once established are not cured by the addition of vitamin A [6] to the diet.

It has been estimated that the anatomic and physiologic development of the rat in one year is approximately equivalent to that in thirty years in man. This formula—accepted as practically correct—has been employed to determine the effect of deficiency in vitamin A on the susceptibility to infection in children from similar observations made on the rat. An insufficiency of vitamin A in early life not only lowers the resistance to infection at the time,[7] but this tendency continues until the child is from 10 to 12 years of age, even when the diet subsequently is normal in every particular. This statement can be applied with particular emphasis to colds, sinusitis and otitis media, as it is known that the nasal and tympanic mucosa are early and frequently infected.

There seems to be two distinct clinical types of vitamin A deficiency in man; the direct and the latent. The former type is seen in patients with xerophthalmia, "catarrhal discharges" and aphonia who have received an insufficient amount of vitamin A for weeks. Cases in this group have been reported from Russia,[8] from Japan, from Manchuria during famine and from Denmark [9] among the rural population when butter is exported in large amounts. Only one case,[4] that of an infant, with observations post mortem, has been recorded in the United States. The pathologic condition and the symptoms observed in such patients are found to be quite similar to, or the same as, those produced on the experimentally fed rat.

In a rat in which A avitaminosis develops, metaplastic and inflammatory reactions in the nasal epithelium precede those in the ocular epithelium, but the eye is normal in a rat that has recovered from this deficiency, while the nose and ear are not. The latent type is found when the nasal symptoms of avitaminosis are present without any other indication, and may occur in either of the aforementioned stages. This condition presents essentially the same nasal symptoms as infection, with which it may readily be confused. Infants and small children are

6. Daniels, A. L.; Armstrong, M. E., and Hutton, M. K.: Nasal Sinusitis Produced by Diets Deficient in Fat-Soluble A Vitamin, J. A. M. A. **81**:828 (Sept. 8) 1923.

7. Sherman, H. C., and Burtis, M. P.: Vitamin-A in Relation to Growth and to Subsequent Susceptibility to Infection, Proc. Soc. Exper. Biol. & Med. **25**:649 (May) 1928.

8. McCollum and Simmonds (footnote 1, p. 159).

9. Block, C. E.: Further Clinical Investigations into Diseases Arising in Consequence of Deficiency in Fat Soluble A Factor, Am. J. Dis. Child. **28**:659 (Dec.) 1924; Decline in Immunity as Symptoms Due to Deficiency in A-Vitamine and C-Vitamine, Acta paediat. (supp. 2) **7**:61, 1928. Widmark, E.: Vitamin-A Deficiency in Denmark and Its Results, Lancet **1**:1206 (June 14) 1924.

regarded as the only section of the population in the United States that can be affected to any extent by this deficiency. Many observers are convinced that in this way an insufficiency of vitamin A is an element not to be ignored in common colds and sinusitis in infancy,[10] while others consider it of etiologic importance in atrophic rhinitis,[11] atrophic pharyngitis and vasomotor rhinitis in older persons. It should be added that the latter view is in dispute, as different conclusions [12] have been reached by others.

The effect of a deficiency in vitamin A on the mechanism of immunity has been repeatedly investigated. The consensus [13] is that no serologic investigation has demonstrated a break in immunity. At the same time, extensive infections of the nasal and tympanic mucosae are present, with gram-negative cocci, streptococci and staphylococci, particularly the chromatogen-6.[14] It has been concluded that the general resistance to infection remains intact with a breakdown in local defenses.[15]

The use of vitamin A in preventing infections of the upper respiratory tract has been advised by many authors. The value consists of its capacity to increase the local resistance to bacterial invasion. In addition to its nutritional properties, some have claimed for it a specifically curative effect [16] on conditions due to Streptococcus hemolyticus. This action has been denied by others,[17] who present evidence in support of their contention.

The minimal amount of vitamin A required for growth and for an unmarred epithelium has not been determined for the human being. It

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10. Dean, L. W.: Paranasal Sinus Disease in Infants and Young Children, J. A. M. A. **85**:319 (Aug. 1) 1925. Dutcher, R. A.: Vitamins in Human and Animal Nutrition, Ann. Otol., Rhin. & Laryng. **38**:593 (Sept.) 1929.

11. Glassheib, A.: Genuine Ozaena as an Avitaminosis, Monatschr. f. Ohrenh. **65**:338 (March) 1931.

12. Fleischmann, O.: Is Ozaena an Avitaminosis? Arch. f. Ohren-, Nasen- u. Kehlkopf. **162**:87 (July) 1930.

13. Werkman, C. H.: Immunologic Significance of Vitamins, J. Infect. Dis. **32**:247, 1923.

14. Shurly, B. R., and Turner, R. G.: Infection of Accessory Sinuses and Upper Respiratory Tract in Vitamin A Deficiency, J. A. M. A. **94**:539 (Feb. 22) 1929. Turner, R. G.; Anderson, D. E., and Loew, E. R.: Bacteria of Upper Respiratory Tract and Middle Ear of Albino Rats Deprived of Vitamin-A, J. Infect. Dis. **46**:328 (April) 1930.

15. Cramer, W., and Kingsbury, A. N.: Local and General Defences Against Infections and Effect on Them of Vitamin-Deficiency, Brit. J. Exper. Path. **5**:300 (Oct.) 1924.

16. Green, H. N., and Mallanby, E.: Vitamin-A as an Anti-Infective Agent, Brit. M. J. **2**:691 (Oct. 20) 1928; Vitamin-A as an Anti-Infective Agent; Its Use in Treatment of Puerperal Septicaemia; Preliminary Communication, Brit. M. J. **1**:984 (June 1) 1929.

17. Burton, A. H. G., and Balmain, A. K.: Vitamin-A and Streptococcal Antitoxic Immunity, Lancet **1**:1063 (May 17) 1930.

seems to be established experimentally that epithelial changes precede those in bone. It has been shown that the vitamin A content in cow's milk while the cattle are given feed in winter is materially less than when they are grazing in summer. Milk is the main source of this vitamin in the diets of many babies. A lowered vitamin intake at an age requiring the maximal amount is regarded by many as not only a possibility, but a probability. No one has said that this avitaminosis occurs frequently in this country, and no one has denied its relative rarity among children on inadequate diets.

Vitamin D.—Vitamin D, among its other characteristics, has the property of controlling the mineral constituents of bone. Clinically, the diet deficient in vitamin D with diminished sunlight causes rickets, and also seems to be associated with the appearance of osteomalacia [18] and osteoporosis. The specific changes in the tissue occur in the vesicular cells of hyaline cartilage and in the preosteal or osteoid tissue, and result in a relatively soft and irregular zone of calcification. This process consists of a disturbance in the mechanism of calcium deposition during the growth of bone. Deficiency in vitamin D is not the only factor in this process, for the absolute and relative amounts of calcium and phosphorus [19] in the food exert a profound effect on the formation of bone.

An investigation [20] has been made with a diet deficient in fat soluble vitamins (A and D) to determine its effect on the osseous labyrinth. The result of this has been recorded as definite rachitic changes in the labyrinthine capsule. These changes consist of softening of the bony labyrinth, bowing of the foot-plate of the stapes, involvement of Cozzolino's zone more often than of the posterior margin of the oval window, partial ankylosis of the stapes and labyrinthine symptoms. The effects of this deficiency on the labyrinth are analogous to the lesions found in otosclerosis. However, this conclusion has not passed unchallenged. The osseous labyrinth [21] in the rat with a deficiency of vitamins A and D is reported to be unchanged in hardness, calcium content and auditory functions.

Vitamin B and Vitamin G.—Vitamin B has been shown to contain at least two factors,[22] the thermolabile, or antineuritic, and the thermostable, or pellagra-preventive. Further evidence has been submitted that

18. McCollum and Simmonds (footnote 1, p. 285).

19. McCollum, E. V.: Pathologic Effects of Lack of Vitamin-A and Anti-Rachitic Element, J. A. M. A. **81**:894 (Sept. 15) 1923.

20. Kaufman, A. B.; Creekmur, F., and Schultz, O. T.: Changes in Temporal Bones in Experimental Rickets, J. A. M. A. **80**:681 (March 10) 1923.

21. Barlow, R. A.: Does Vitamin Deficient Diet Cause Deafness? Results of Animal Experimentation, Laryngoscope **37**:640 (Sept.) 1927.

22. Goldberger, J.; Wheeler, G. A.; Lillie, R. D., and Rogers, L. M.: A Further Study of Butter, Fresh Beef and Yeast as Pellagra Preventative, Pub. Health Rep. **41**:297 (Feb. 19) 1926.

a third factor,[23] one concerned with stimulating growth, is present, but general acceptance of this evidence has not been recorded. The antineuritic factor is now called vitamin B and the pellagra-preventive, vitamin G by the American biologic chemists.

The specific changes in the tissues in deficiency of vitamin B seem to be atrophy of lymphoid tissue and lymphopenia. More important than this is the part that such deficiency plays in cell metabolism.[24] It is said to cause vacuolation and disintegration of the medullary sheath [25] of nervus acusticus. The changes in the ganglion cells of the nervus vestibularis are much greater than those in nervus labyrinthicus. The pathologic and clinical changes in the laboratory species on a diet deficient in vitamin G approximate the lesions of pellagra in man.

The therapeutic [26] use of vitamin B has been recommended in aphthous ulcers, in gingivitis and in Vincent's angina.

EXPERIMENTAL INVESTIGATION

The experimental investigation was carried out with seventy albino rats. At approximately 1 month of age, the following number of rats were placed on diets deficient in the vitamin specified: vitamin A, 16; vitamin B, 10; vitamin B complex (B and G), 12; vitamin D, 10 in a dark room and 8 in subdued daylight, and vitamin G, 10; 4 were kept as controls on a balanced diet. These diets were continued for from three to five months, when the surviving animals were placed on normal feedings. After four additional months on a complete diet, selected animals were killed. Microscopic sections of the nose, ears, pharynx, larynx, trachea and ribs were examined in thirty-seven. The direct effect of the avitaminosis on the upper respiratory tract and the ear could be studied in the animals killed or dying while on the faulty diets. The latent results could be also observed in those given normal food for four months. In this way it was hoped that an estimate might be formed of the direct and latent results of avitaminosis in man.

A constant and striking characteristic of deficiency in each vitamin observed in these rats was the variable susceptibility. One rat died of the effect before another in the same group was more than slightly affected. The development of symptoms was inconstant, and seemingly wholly dependent on the individual animal. Young rats were far more sensitive than the older ones to a diet inadequate in vitamins.

RESULTS OF AVITAMOSIS IN RATS

Vitamin A.—Xerophthalmia was observed in twelve rats on a diet deficient in vitamin A. A nasal discharge was present in thirteen.

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23. Reader, V.: Second Thermolabile Water Soluble Accessory Factor Necessary for Nutrition of Rat (B_3 in Yeast Extract), Biochem. J. **33**:689, 1929.

24. Cowgill, C. R.: Physiologic and Biochemic Aspects of Deficiencies, Ann. Int. Med. **3**:230 (Sept.) 1929.

25. Ono, T.: Experimental Histopathlogic Studies of the Ear with B- and C-Aphtho-Ulcerative) Stomatitis and Herpes Labialis, Am. J. Dis. Child. **26**:309 (Oct.) 1923.

26. Gerstenberger, H. J.: Etiology and Treatment of Herpetic (Aphthous and Avitaminosis, Tr. Jap. Path. Soc. **18**:172, 1928.

Anosmia was evident in many after they became blind. The failure of several to squeak after their tails were pinched indicated aphonia. Impaired hearing was repeatedly demonstrated. At necropsy, abscesses of the salivary glands and middle ears were common. The nose and trachea were not opened at the postmortem examination.

A detailed account of the histopathologic changes in the olfactory and respiratory mucosa of rats killed or dying while on a diet deficient in vitamin A is not necessary, as there are already several excellent descriptions. Briefly, the salient features are the presence of numerous metaplastic foci in the deeper layers of epithelium during the stage of avitaminosis and, later the extension of these foci to the surface as

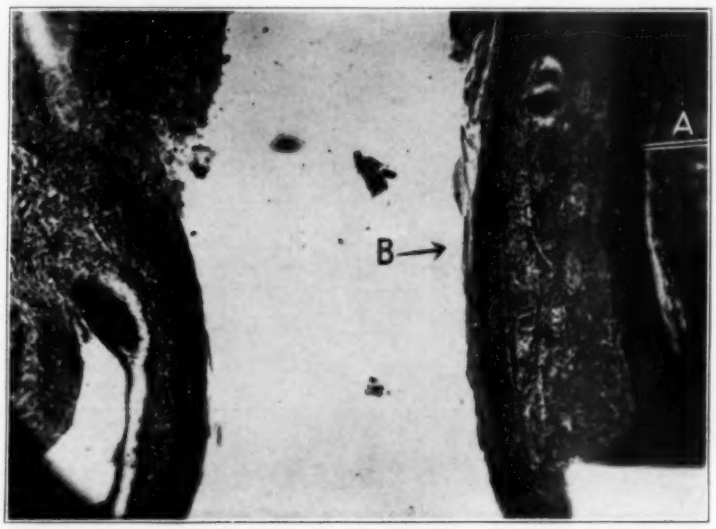

Fig. 2.—Nasal mucosa of a rat on a diet deficient in vitamin A. *A* indicates the septum; *B*, metaplasia of cylindric epithelium to stratified squamous with keratinization; × 100.

stratified squamous epithelium (figs. 2, 3 and 4), with subsequent keratinization. A tenacious mucopurulent discharge with detritus is present in the nasal meatuses, and spindle cells, round cells and polymorphonuclear leukocytes appear in large numbers in the edematous mucosa. The respiratory mucosa of the nose is affected before the olfactory mucosa. The turbinate bones are uniformly thinner than normal and increased activity of osteoclasts is evident in the later stages. In the upper part of the trachea, the process is the same as that in the nose, except for the hyperplasia (fig. 5) of stratified squamous epithelium. The stratified squamous epithelium in the pharynx is thicker than normal. The tympanic mucosa (fig. 6) shows stratified squamous epithelium and inflammation.

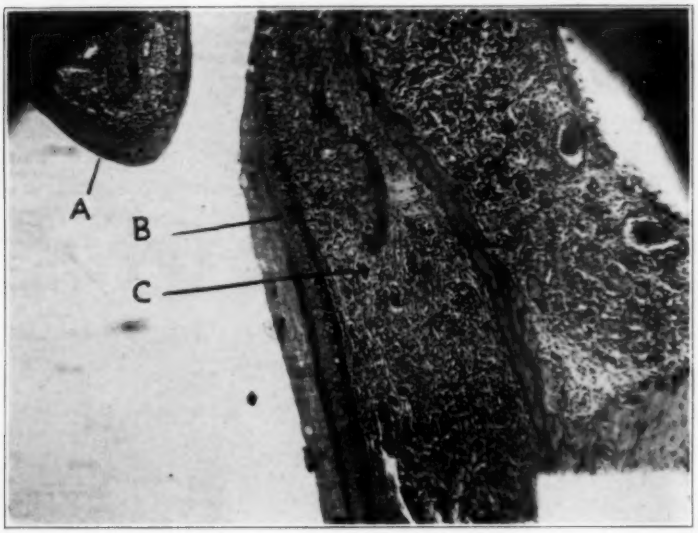

Fig. 3.—Nasal mucosa of a rat on a diet deficient in vitamin A. *A* indicates tip of middle turbinate; *B*, stratified squamous epithelium; *C*, subepithelium, showing round cells and leukocytes; × 100.

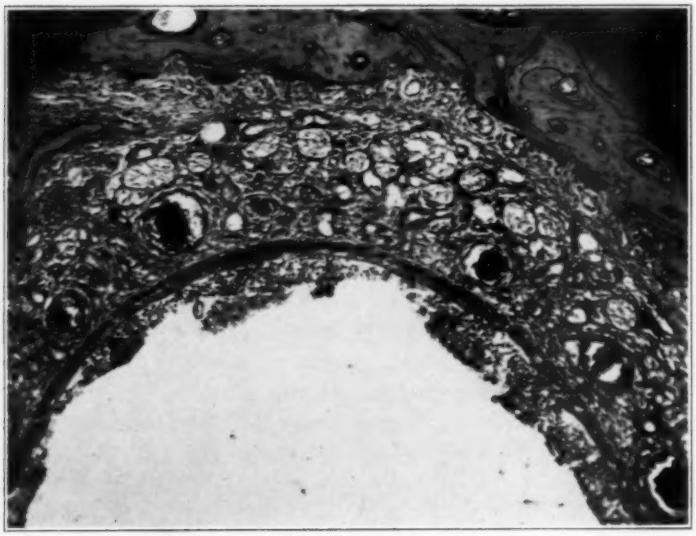

Fig. 4.—Olfactory nasal mucosa of a rat on a diet deficient in vitamin A. The metaplasia of cylindric epithelium to stratified squamous explains the anosmia. The mucous and serous glands are enlarged; × 100.

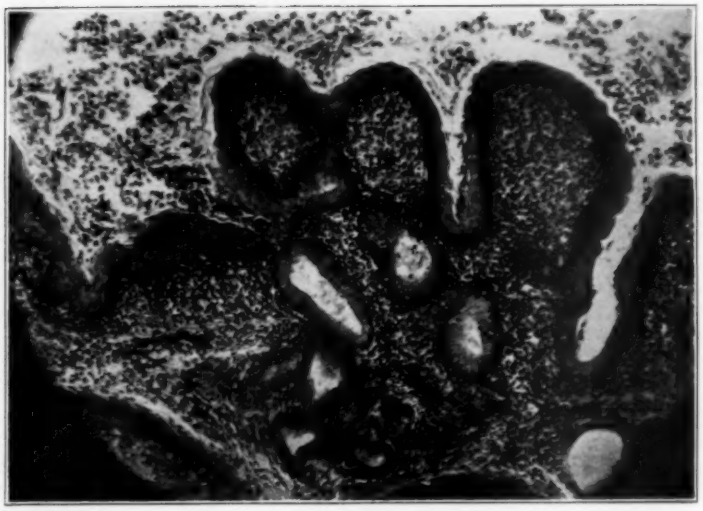

Fig. 5.—Tracheal mucosa of a rat on a diet deficient in vitamin A. There is hyperplasia of the mucosa with stratified squamous epithelium. Note the resemblance of the condition to that seen in figure 1; × 100.

Fig. 6.—Aural mucosa of a rat on a diet deficient in vitamin A. Metaplasia of the cylindric epithelium to stratified squamous epithelium with keratinization. The process is identical with that found in the nose. *A* indicates bone; *B*, mucosa; *C*, stratified squamous epithelium with keratinization; *D*, pus and detritus; × 100.

In the rat on a normal diet for four months after the avitaminosis, the nasal mucosa, while not normal, shows no stratified squamous epithelium. The cylindric cells are usually ciliated and flatter than normal with clearer cytoplasm and relatively larger nuclei. The cells are occasionally clustered as villi. The subepithelial tissue has an increase in fibrous tissue and round cells, and the blood vessel walls are thicker. The turbinate bones are normal.

Vitamin D.—The rats on a diet deficient in vitamin D had a shambling gait, difficulty in rising, bow legs and rosary ribs. Nasal discharge and impaired hearing were not observed. No differences in the ears, nose and throat were found between the rats kept in the dark

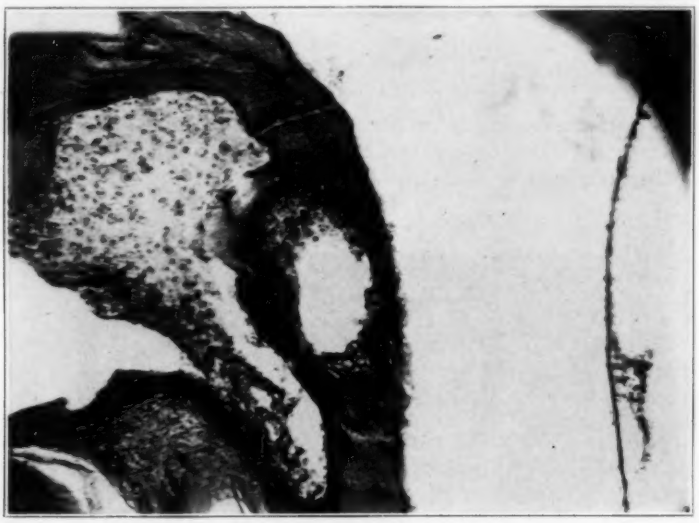

Fig. 7.—Vertical section through stapediovestibular joint in a rat on a diet deficient in vitamin D. The cells of elastic cartilage show no changes. The bending of the foot-plate of the stapes is within normal variation; × 100.

room and those kept in subdued daylight. The nasal mucosa did not show metaplasia or hyperplasia. Infection was not present, but the round cells and polymorphonuclear leukocytes were diminished. The mucous membranes of the pharynx, larynx and trachea were normal. The turbinate bones and septums showed no rachitic changes.

The temporal bones were so soft that care was necessary not to dislodge the labyrinths. A specific change in the tissue analogous to that at the epiphyseal line was not seen in the labyrinthine capsule. No abnormality was observed in the elastic cartilage of the stapediovestibular (fig. 7) joint. The cartilaginous cells next to the bone had the same appearance as those in the controls. Any bending in the foot-plate of

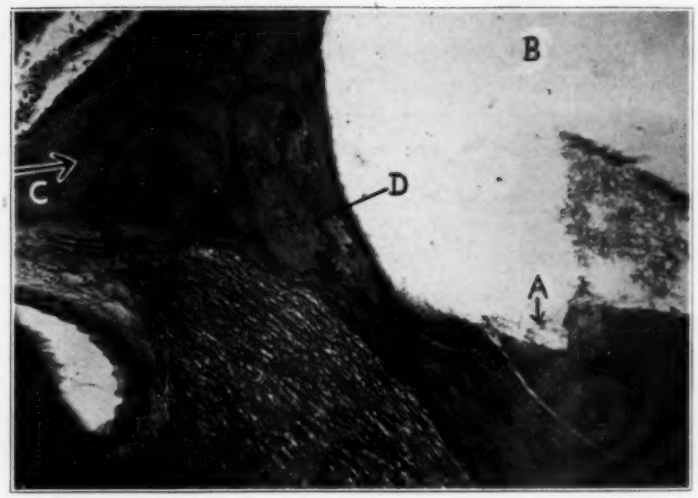

Fig. 8.—Horizontal section through the posterior part of the stapediovestibular joint in a rat on a diet deficient in vitamin D. *A* indicates the posterior portion of the foot-plate of the stapes; *B*, the vestibule; *C*, an area of spindle cells in the middle layer, and *D*, an area showing enlarged preosteal cartilaginous cells in the middle layer; × 100.

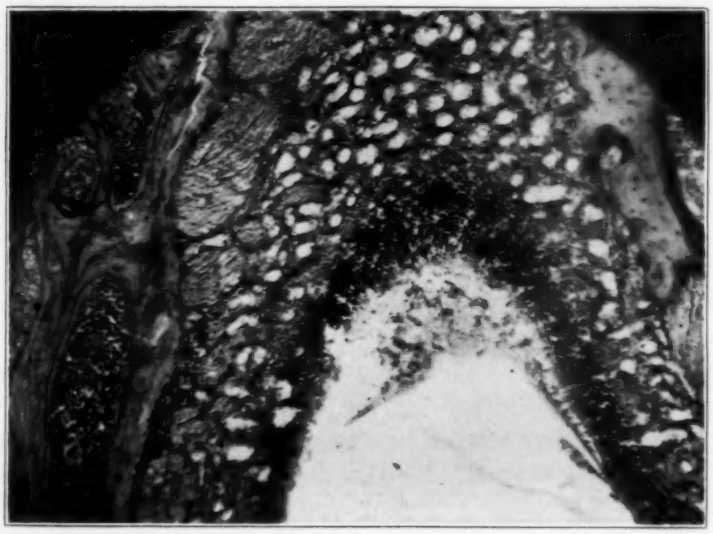

Fig. 9.—Vertical section through the posterior ethmoidal region in a rat on a diet deficient in vitamin B. The polycystic appearance of the mucosa is due to dilatation of the acini of Bowman's glands; × 100. Hypertrophy of the mucous glands is noted.

the stapes appeared to be within the variation of normal. Some of the preosteal cartilaginous cells (fig. 8) in the middle layer were larger and more spindle-shaped than those in the controls. In the rats on a normal diet for four months after the avitaminosis, no otosclerotic foci were observed in the middle layers or at the upper anterior edge of the fenestra vestibuli.

Vitamin B.—The characteristic changes in the tissue of rats on a diet deficient in vitamin B (fig. 9) is the polycystic appearance of the nasal mucosa in the upper posterior ethmoidal region. This is due to the dilatation of the lumen of Bowman's glands, with the flattening of the lining membrane of the cuboidal cells. The round cells and the leukocytes in the mucosa are diminished. The respiratory mucous membrane of the nose is not affected. The mucous membrane of the tympanum is normal, except that the leukocytes and round cells are less numerous.

Vitamin G.—No abnormalities in the ear, nose and throat typical of deficiency in vitamin G are noted.

CLINICAL APPLICATION

Vitamin A.—Clinical application of experimental data can be made with accuracy and confidence, for the symptoms and pathologic conditions of A avitaminosis in the rat are prototypes of those found in man. Vitamin A has been shown to have the property of maintaining the integrity of the epithelium in the upper respiratory tract and middle ear. The clinical application of this fact is that the direct effect of an inadequate diet must be added to infection and allergy as a cause of such conditions as frequent colds, chronic sinusitis and recurrent otitis media in infants and children. The avitaminosis may be distinguished from bacterial infection in chronic conditions of the nose by the presence of a bilateral pansinusitis in underdeveloped children with anorexia and of a tenacious mucopurulent discharge that is but little diminished by local treatment. The borderline between these diseases is wide, and here the differential diagnosis is impossible with present knowledge. Recurrent otitis media, particularly when associated with a sinusitis similar to that described, is suggestive of this deficiency.

As a later result of the avitaminosis, the nasal mucosa of the rat requires considerably more time to recover than does the eye. Eventually, the metaplasia disappears, though the epithelium is not normal. The clinical application of this fact is that a deficient diet in infancy and early childhood affects the nasal mucous membrane for several years, but not beyond puberty. The symptoms are anosmia, thick mucopurulent discharge adherent to an atrophic mucosa, atrophied turbinate bones, no odor and subnormal physical development. The histopatho-

logic picture is stratified squamous epithelium, thickened blood vessel walls, atrophied turbinals and increase in fibroblasts, round cells and leukocytes in the subepithelium. This syndrome is characteristic of atrophic rhinitis without odor in children.

The status of avitaminosis as an etiologic factor in atrophic rhinitis must be held *sub judice* at present. There are too many facts pointing to this deficiency as a probable cause of atrophic rhinitis for it to be ignored, but not enough proof can be presented to be conclusive. Thus, anosmia, chronic nasal mucopurulent discharge, crusts in the pharynx, atrophic mucosa with stratified squamous epithelium; fibroblasts, round cells, leukocytes and thickened blood vessel walls in the subepithelium, and atrophied turbinate bones are found in the rat recovering from avitaminosis. These observations are identical with those in atrophic rhinitis. Also, the more frequent occurrence of the condition in patients seen in the clinic than in those seen in private practice is suggestive of a dietary inadequacy. On the other hand, if avitaminosis is the cause the age of onset should be early childhood instead of late childhood and early adult life as it usually is. All the data now available indicate that the results of a deficiency in infancy cannot extend beyond puberty. The odor of ozena has not been detected in the rat. Further experimental investigation is necessary to determine the extension and recession of metaplastic changes. However, if the assumption that some persons are very susceptible to vitamin deficiency is correct, every requirement for avitaminosis as a cause of atrophic rhinitis has been met.

The treatment of acute infectious diseases of the ear, nose and throat by vitamin A in the form of cod liver oil has been employed frequently, without any specific curative action on the invading bacteria being noted. The value of vitamin therapy in the direct type of sinusitis is difficult to estimate, because the differential diagnosis is so uncertain. Vitamin A as an adjunct to local treatment has seemed to be of considerable benefit in sinusitis in many children, for with it recoveries are more prompt and recurrences less frequent. Two children with atrophic rhinitis without odor have been treated with vitamin A in the form of cod liver oil and butter fat for from eight to ten months during the past year, with improvement. These results are not conclusive, but suggest that in chronic infections vitamin A improves the nutrition of the nasal mucosa. This vitamin has been of no benefit in the treatment of vasomotor rhinitis and postoperative atrophic rhinitis.

The incidence of acute infections in the ear, nose and throat among the infants and children at Houston was sharply reduced two years ago after the routine use of vitamin A was begun. It is apparently of great value in the prevention of infections of the upper respiratory tract and ears. However, its employment was started about the time that a severe epidemic of influenza was on the wane, so that the seeming prophylaxis

may be a coincidence. During a time that vitamin A has been used, hypertrophy of the tonsils has been encountered less frequently in children on this regimen. Enlarged tonsils have been observed to decrease in size. The final appraisal of the prophylactic value of vitamin A should be deferred for several years until the epidemic cycle has been completed. In the meantime, its widespread and routine use is to be encouraged, as only in this way can an estimate be formed of its worth as a preventive of nasal infection and of tonsillar hypertrophy.

Vitamin B.—A syndrome has been identified with deficiency of vitamin B. The complaint is a slight but frequent postnasal discharge. Mucus is expectorated at short intervals. Sneezing is occasional, but not in bouts. There is no headache. Young women are most often affected, and constipation is uncommon. On anterior examination the nasal mucosa is found to be practically normal, except over the middle turbinate. Posteriorly, the tips of the turbinate have a smooth, moist, creamy-white and slightly thickened appearance. Mucus is seen in various locations, but not regularly in any one place. No mucopus is found. The leukocyte and differential counts are normal. The condition is to be distinguished from allergic reactions and from hyperplastic ethmoiditis. In the former, conjunctivitis, profuse nasal discharge, uniform swellings of turbinates, grayish-white mucosa, paroxysmal sneezing and eosinophilia are present. In the latter, the mucosa of the middle turbinate is indurated, hyperemic and frequently redundant.

Five patients presenting this group of symptoms have been given vitamins B and G in the form of brewer's yeast during the past year. As a control, laxatives were given to five patients without any affect on the nasal discharge before the vitamin was employed. The postnasal discharge is decreased about 75 per cent in three days, but it does not disappear. Brewer's yeast was used because it was not known which vitamin deficiency was responsible until the completion of the animal experiments. These implicated vitamin B and cleared vitamin G. The employment of yeast was continued for the sake of uniform comparisons. The number of cases is small for any conclusion, but so far as they go they indicate a deficiency in vitamin B of the nasal mucosa and its remedy.

CONCLUSIONS

Vitamin A is necessary to maintain the nutrition of the nasal, aural and tracheal epithelia. The nasal mucosa is affected before and recovers after the ocular mucosa. Eventually, the metaplastic and inflammatory changes of the deficiency are cured by a normal diet. Prophylactically, vitamin A seems to increase the resistance to upper respiratory infection in children. Therapeutically, its chief value is to improve the nutrition of the nasal and aural mucous membranes in acute and chronic infections.

While this avitaminosis may be an etiologic factor in atrophic rhinitis, further investigation is required before deciding its status.

A diet deficient in vitamin D has no effect on the nasal, aural and tracheal mucosa. It produces no lesion in the osseous labyrinth in the rat that resembles otosclerosis in the human being.

Deficiency in vitamin B is identified with a definite nasal syndrome. This consists essentially of a postnasal mucous discharge, and the posterior tips of the middle turbinates are smooth, moist, creamy white and slightly thickened.

Deficiency in vitamin G has no effect on the ear, nose or throat.

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CARCINOMA OF THE LARYNX

CLINICAL AND PATHOLOGIC ASPECTS

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PHILADELPHIA

Laryngologists are unanimous in their opinion that early diagnosis and complete surgical extirpation are essential in the successful treatment of cancer of the larynx. Careful selection of patients, adequate preoperative preparation, improved methods of anesthesia, perfection of surgical technic and proper postoperative care have contributed enormously to the safety of surgical treatment so that the operative mortality rate has reached an incredibly low level. To reduce further the mortality rate of cancer of the larynx it will be necessary to improve diagnostic methods, particularly from the standpoint of early diagnosis, and to add to the knowledge regarding degrees of malignancy and metastasis.

It has been estimated that approximately 4 per cent of all malignant tumors occur in the larynx, and that about 98 per cent of these belong to the squamous cell variety. Study of large groups of reported cases of laryngeal carcinoma indicates that only between one third and one fourth of these are seen sufficiently early to permit of some form of surgical treatment. While many of the patients with advanced cases do not consult a physician until their condition has become hopeless, a considerable number, unfortunately, have been under observation and treatment for many months, without a diagnosis of cancer having been made. One would believe that the diagnosis of laryngeal cancer is difficult or that its symptoms are not well understood by the medical profession.

Although it is not our intention to discuss mirror laryngoscopy as a means of diagnosis, it must be emphasized that this should be the first procedure employed when symptoms of laryngeal disease are present. Without this there is little need of discussing refinements in diagnosis, since one must first recognize the presence of laryngeal disease before proceeding with its differential diagnosis.

Read at a meeting of the Triological Society, Atlantic City, N. J., May 23, 1932.

From the Bronchoscopic and Pathological Departments and Tumor Clinic, Jefferson Medical College Hospital.

DIAGNOSIS

If cancer of the larynx is suspected, the case should be promptly brought to a final conclusion by utilization of all of the means at one's disposal. While the expectancy of life in the average case of untreated cancer is approximately two years, this can be added to only if the diagnosis is made early and adequate treatment is promptly instituted. Surgical treatment, to be effective, must be carried out during the early stages of the disease, a period best reckoned in months, not years.

Precancerous Lesions.—In chronic inflammatory lesions of the larynx in which there is marked hyperplasia of the epithelium in an effort at repair, this hyperplasia and irregularity frequently suggest early malignant changes. The term "precancerous" has been applied to this type of lesion by many writers. There may be some justification for its use by the laryngologist when he is considering certain benign lesions that have undergone malignant changes. In the histologic examination of tissue in a number of these cases it was impossible to predict with any degree of certainty in which cancer would develop and in which recovery would occur. In the following up of many of these cases a majority were found to clear up under vocal rest combined with other treatment. In a small group cancer developed. The following case reports are illustrative.

CASE 1.—*Suggestive malignant growth of the vocal cord which cleared up under vocal rest.*

A man, aged 63, a music teacher, complained of hoarseness of seven months' duration. A small papillary growth with superficial ulceration partially covered with whitish exudate was found, involving the left cord at the junction of the middle and anterior thirds. An inferential diagnosis of cancer was made, and a specimen of tissue was removed by direct laryngoscopy. Plans were made to proceed with laryngofissure if the histologic diagnosis proved to be cancer. The histologic findings were a markedly thickened epithelial surface with hyperkeratosis, hyperplasia and irregularity of the deeper epithelial layers and an inflammatory reaction of the subepithelial tissue. The changes in the epithelium were suggestive of early malignancy, but examination of many sections from the tissue failed to reveal definite infiltration by the epithelium (fig. 1). Operation was deferred, and the patient was kept under observation on a regimen of vocal rest. The lesion healed promptly, and the larynx now is normal, fifteen months after the biopsy.

CASE 2.—*Suggestive malignant growth of the vocal cord which ultimately became cancerous.*

A man, aged 57, a salesman, had been hoarse for five or six years. The hoarseness became progressively worse. In 1924, there was found a small nodule on the middle third of the right vocal cord. It was not ulcerated. The nodule was removed by direct laryngoscopy for biopsy. Histologic examination revealed marked thickening and irregularity of the surface epithelium with pegs of epithelium extending into the tissue beneath and a diffuse inflammatory reaction. Many sections were examined, but definite evidence of malignant change could not be demonstrated

(fig. 2). The patient was kept under observation on a regimen of modified vocal rest. Three years later there was noted a beginning ulceration on the right vocal cord corresponding to the area previously involved. A specimen was again removed, and this proved to be a squamous cell carcinoma (fig. 3). Laryngofissure was promptly employed. The patient made a satisfactory recovery. There has been no recurrence of the growth to the present time, a period of over five years.

As it is impossible to determine histologically which of these lesions will ultimately become malignant, it does not seem practical to employ this term. Lesions may be suggestive of malignancy and should be so

Fig. 1.—Photomicrograph (× 57) from a lesion of the vocal cord illustrating localized pachydermia, with inflammatory reaction and marked hyperplasia and irregularity of the epithelium, suggestive of malignant changes. This lesion cleared up without developing into carcinoma.

reported. The laryngologist can then be guided in his decision regarding operation. Although the patient in case 2 has had no recurrence after more than five years, there is a question whether laryngofissure should not have been performed in 1924 on the basis of the suspicious histologic report.

Syphilis.—The presence of a positive Wassermann reaction frequently lulls the physician and the patient into a false state of security. Valuable time is often wasted in the prolonged use of antisyphilitic

treatment. In a syphilitic lesion, appropriate treatment should be followed by rapid improvement. In cases of cancer there are often observed slight changes in the lesion following antisyphilitic treatment, and this is promptly interpreted as unmistakable evidence that the lesion is syphilitic. If the improvement is not prompt and continued, the diagnosis of syphilis should be considered as inconclusive, and biopsy performed. In our experience the concomitance of cancer and syphilis of the larynx has been rare; in fact, the coexistence of systemic

Fig. 2.—Photomicrograph (× 92) from a lesion in the larynx which was histologically similar to the lesion illustrated in figure 1. Areas in this lesion were very suggestive of a malignant growth, but definite evidence of infiltration by the epithelium could not be demonstrated in any of the sections from this specimen. Carcinoma developed three years later, as illustrated in figure 3.

syphilis and cancer of the larynx has occurred in 4.5 per cent of two hundred cases. The diagnosis of syphilis was based on a strongly positive Wassermann reaction. This compares favorably with the incidence of syphilis in the general population.

Tuberculosis.—A typical tuberculous lesion of the larynx in the presence of demonstrable pulmonary tuberculosis should offer no diag-

nostic difficulties. In atypical lesions, if the pulmonary disease is quiescent a biopsy should be made. Tuberculosis and cancer may be found concurrently in the larynx. The reported cases are few. Cancer of the larynx may occur in a patient with pulmonary tuberculosis. Careful laryngeal examination and biopsy should aid in arriving at a final conclusion. While biopsy is considered by some as being a hazardous procedure in tuberculosis, experience in a considerable number of cases has not convinced us that this is a fact.

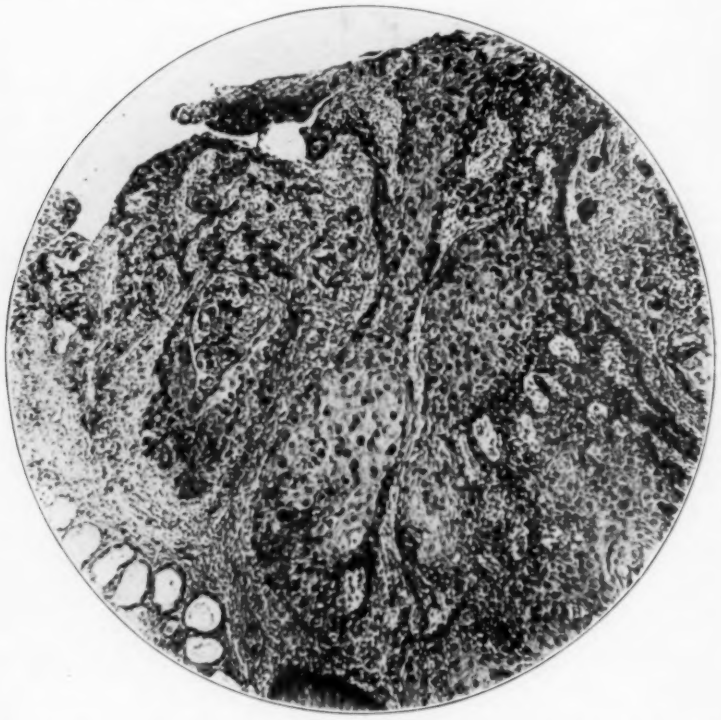

Fig. 3.—Photomicrograph (× 92) of section illustrating definite carcinoma which developed at the site of the previous lesion, illustrated in figure 2.

BIOPSY

The practice of removing a portion of tissue from the larynx for diagnostic purposes has always been a subject of controversy. In the typical case of cancer a confirmatory microscopic study may appear unnecessary. The question, however, arises whether one is justified in carrying out a radical form of treatment without first securing confirmation of the diagnosis. Many objections to biopsy are offered. Chief among these are failure to secure corroboration of the clinical diagnosis and the danger of facilitating dissemination of the growth. The pathologist can give an opinion only on the tissue submitted to him

and not on the entire growth. Many of the criticisms of preoperative histologic examinations are due to failure to secure a representative specimen of tissue from the lesion, lack of proper technic in the preparation of the small fragments of tissue submitted and inexperience on the part of the pathologist in the microscopic examination of lesions of the larynx. If a representative specimen of tissue has been submitted, the report should carry much weight. It must be considered in conjunction with the history, clinical appearances and collateral studies.

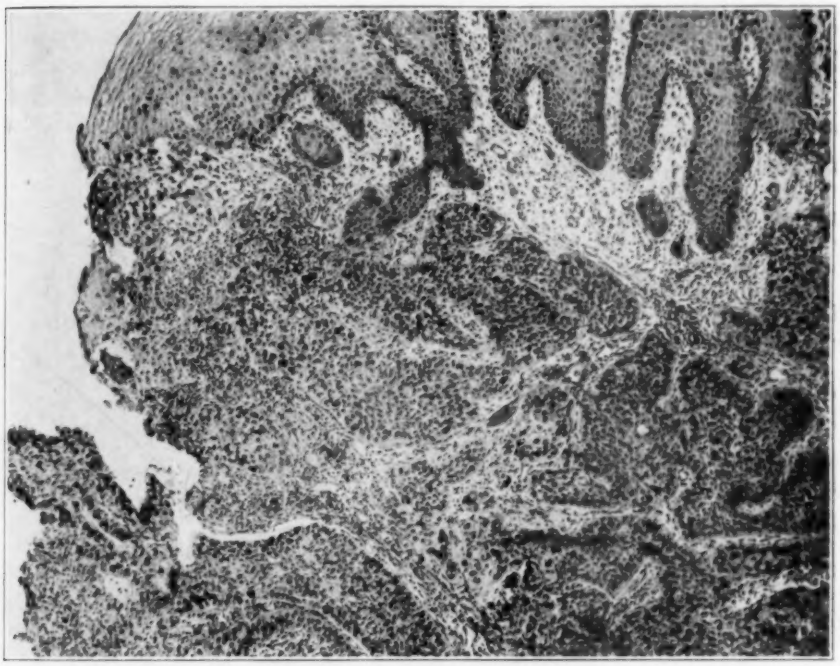

Fig. 4.—Photomicrograph (× 57) of biopsy specimen of advanced carcinoma of the larynx illustrating an ideal specimen taken from the margin of the lesion. The transition can be observed from the normal squamous epithelium of the mucosa to the masses of undifferentiated cells infiltrating the deeper structures.

An adequate specimen should consist of a portion of the growth including an edge of the normal tissue (fig. 4).

Care must be exercised in securing a portion of the base of the lesion as well as a part of the normal tissue. Frequently, in inflamed and ulcerated lesions and in papillary growths, specimens removed from their surface may not contain definitely malignant tissue while examination of tissue from the base of the lesion will reveal cancer. With the aid of the Jackson laryngoscope an adequate specimen from

the desired location can be secured without difficulty. If the report is negative in spite of the suggestive clinical appearances, another specimen should be secured.

The histologic diagnosis of cancer is simple in the advanced case, while in the early case in which the lesion is small and complicated by inflammatory changes it is frequently difficult to differentiate between a malignant lesion and an inflammatory one in which there is marked hyperplasia of the squamous epithelium. It is therefore imperative in preparing the specimen for histologic study to employ a technical method that will give thin and well stained sections. The frozen section method is not applicable to or sufficiently reliable for the examination of small pieces of tissue of this type. We use a rapid paraffin method,[1] which gives excellent results, and a reliable report on the tissue may be submitted within eighteen hours. The most difficult and

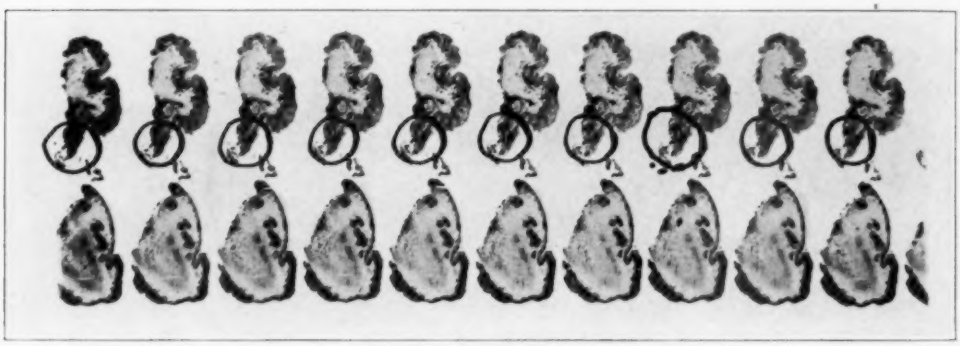

Fig. 5.—Photomicrograph of a slide containing twenty sections from a small biopsy specimen of the larynx, illustrating the method of cutting serial sections and mounting by a rapid (eighteen hour) paraffin method.

important part of the procedure is the removal of the specimen for study. If a small representative piece of tissue is removed, preferably from the margin of the lesion in order to include normal tissue as well as the growth, and is properly oriented and prepared for histologic study by a rapid paraffin technic, by the cutting of serial sections (fig. 5) and examination of a number of sections from different levels in the tissue, a reliable report may be submitted (fig. 6).

Dissemination of Cancer by Biopsy.—In a recent thorough review of the subject of biopsy of tumors by Hellwig,[2] it was pointed out that there remain some objections to biopsies on neoplasms, but it seems that the only class of growths in which the objection is well grounded

1. Crawford, B. L.: Proc. Path. Soc., Philadelphia **30**:18 (Dec. 12) 1929.
2. Hellwig, C. A.: Biopsy in Tumors, Arch. Path. **13**:607 (April) 1932.

is that of closed tumors, such as bone sarcomas and neoplasms of the mammary gland. The consensus seems to be entirely in favor of biopsies in open lesions. With this view we heartily concur. The objection in the past to biopsies on open lesions seems to have been largely theoretical, as no definite proof has been brought forward to show that actual dissemination of the growth was caused by the biopsy. For a number of years we have made a special effort in the examination of specimens removed at operations and autopsies after biopsy

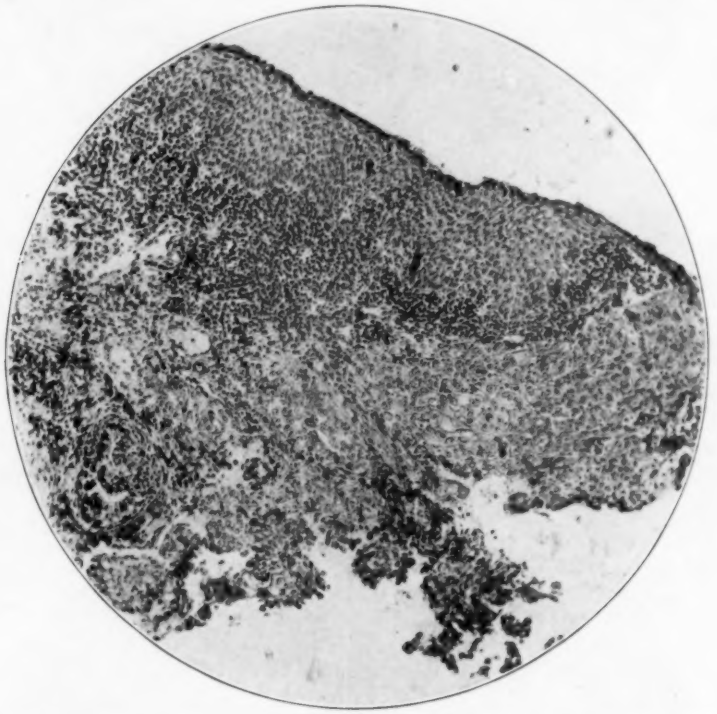

Fig. 6.—Photomicrograph of higher magnification (× 57) of the encircled area shown in figure 5. The area involved in this specimen was very small, but the infiltration of the deeper structures by the undifferentiated epithelium justified the diagnosis of carcinoma.

to determine whether or not there was any evidence of dissemination of the growth following this procedure. Our observations are entirely negative as to the deleterious effects. In one case in which the malignant nature of the small papillary growth of the larynx was not recognized microscopically (fig. 7), the lesion recurred two and one half years later, at which time it was removed by laryngofissure (fig. 8). No evidence of metastasis to the regional lymph nodes was found. There has been no evidence of recurrence for six and one half years. At autopsy

Fig. 7.—Photomicrograph (× 69) from a biopsy specimen from a papillary growth of the larynx, the malignant nature of which was not recognized histologically. The change in the deeper layers of the epithelium should have been recognized as evidence of malignancy. This lesion recurred two and one-half years later with definite infiltration at the site of the previous lesion, as illustrated by figure 8.

Fig. 8.—Photomicrograph (× 92) from the recurrent lesion illustrating the definite infiltration at the site of the previously removed papillary growth.

in a number of cases of inoperable carcinoma of the larynx in which biopsies had been made we were unable to find that the incidence of metastasis was any higher than in cases in which biopsy was not done.

In spite of our conviction regarding metastasis, we have made it a practice to advise patients that we suspected cancer, and have urged biopsy for corroboration of the diagnosis with the understanding that, if the results were positive, the necessary operative procedures should be promptly proceeded with. When the rapid paraffin technic is used, a report may be secured within eighteen hours.

GRADING

Considerable interest has been taken in the histologic grading of tumors according to their degrees of malignancy by Broder's method. It has been shown by a number of workers that there is a definite relationship, especially in lesions from certain locations, between the cell type of the tumor and the prognosis. For several years we have been studying our cases in an effort to determine whether or not any such relationship could be recognized in laryngeal carcinomas. Our studies would seem to indicate that we can differentiate between two extremes, that is, the low grade malignant squamous cell carcinoma in which there is considerable differentiation of the infiltrating epithelium, with much keratin formation (fig. 9), and the high grade anaplastic type, in which the infiltrating epithelium is undifferentiated, with little prickle cell formation, great variety in the size and shape of the cells and their nuclei, and numerous mitotic figures (fig. 10). These groups comprise a relatively small per cent of the cases studied, the majority falling in a large intermediate group. Whether or not the latter group can be subdivided into types 2 and 3, as has been done in malignant lesions of other parts of the body, is a question which can be determined only by further study and a longer follow-up of cases. The conservative view, which would seem to be substantiated by our studies as well as by those of others, is that the cell type of a tumor is important if taken into consideration along with the clinical facts in the case, such as the duration of the disease, the location of the lesion and the age of the patient, in deciding on the treatment and the prognosis.

METASTASIS

One of the most important factors in the treatment of a malignant lesion is the occurrence of metastasis. The fact that carcinoma of the larynx, especially in certain locations, does not metastasize early has been pointed out, but we do not believe that sufficient emphasis has been laid on this important point. In some of our cases, even in the advanced

Fig. 9.—Photomicrograph (× 69) from carcinoma of the larynx illustrating the extent of the differentiation with extensive keratinization in a low grade squamous cell carcinoma.

Fig. 10.—Photomicrograph (× 57) from a carcinoma of the larynx illustrating the undifferentiation of the epithelium in a growth of a high degree of malignancy. This patient died six months following laryngectomy, with extensive metastasis to the lymph nodes.

stages, no clinical evidences of metastasis to the lymph nodes could be demonstrated, and at autopsy in certain fatal cases of cancer of the larynx involvement of even the regional lymph nodes could not be demonstrated. Whether or not there is a definite anatomic reason for this in the lymphatic supply of the larynx seems to be an unsettled point, as the majority of anatomists state that the larynx is abundantly supplied with lymphatics. Another factor which may play a rôle in the lack of early metastasis is the relatively low degree of malignancy in many of these cases. We have observed several cases of carcinoma of the anaplastic type in which extensive metastasis occurred early, whereas in those of low grade malignancy years have elapsed without any evidence of involvement of the regional lymph nodes. This should encourage surgeons to attempt radical removal of these growths in the larynx, as the danger of metastasis seems to be less than is observed in cancer in many other locations.

INDICATIONS FOR OPERATIONS

In determining the type of operation to be employed in a given case the important criteria are the location and extent of the growth. This information is best secured by mirror laryngoscopy, supplemented in certain cases by direct examination and by palpation of the neck. In addition, knowledge concerning the degree of malignancy is important. Laryngofissure, laryngectomy or some of the more radical operations are the only procedures worthy of consideration. Although it may appear that a small tumor of the cord can be entirely removed by endolaryngeal methods (fig. 11), many reports of failure warrant the condemnation of such methods (fig. 12).

ANALYSIS OF CASES

A series of thirty-two cases of cancer of the larynx in which the patients were operated on prior to three years ago was studied. In every case a biopsy was done either to confirm or to establish a diagnosis. No ill effects from this procedure were noted.

In the grading of the tumors six were considered as of low grade malignancy, twenty were intermediate and six were placed in the anaplastic group.

There were six recurrences. Of these, five occurred in cases of the intermediate group and one in the anaplastic group. Because of the high percentage of recurrences in the intermediate group one might question the value of grading from the standpoint of prognosis. We believe, however, that additional study of this question, particularly with regard to the intermediate group, must be carried out.

Fig. 11.—Photomicrograph from a biopsy specimen of a carcinoma of the larynx which includes practically the entire lesion and illustrates that while there is definite infiltration by the epithelium, the lesion is superficial.

Fig. 12.—Photomicrograph (× 57) from a laryngofissure specimen in a case that clinically seemed to be one of an early superficial lesion. Masses of the epithelial cells can be observed infiltrating the deeper structures, including the muscle. This patient is well and free from recurrence seven years after the growth was removed by laryngofissure.

CONCLUSIONS

1. Biopsy should be performed in every case in which laryngeal disease is suspected.

2. A specimen of tissue, properly removed and prepared and studied by a competent pathologist, affords an important aid in diagnosis.

3. No ill effects following biopsy have been demonstrated or observed.

4. The cell type of a tumor should be considered in conjunction with the age of the patient, the location and extent of the lesion and the duration of the disease in determining the form of treatment to be carried out.

5. As a rule, metastasis from laryngeal cancer does not occur early; this should encourage increased efforts at early diagnosis and surgical treatment.

ASYMMETRY OF THE NARES

A POSITIVE DIAGNOSTIC SIGN OR ENTITY ESTABLISHING ANATOMIC DISPLACEMENT OF LOWER END OF CARTILAGINOUS NASAL SEPTUM

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CLEVELAND

DEMONSTRATION OF GEOMETRIC PROPOSITION

When a perpendicular is dropped from the apex of an isosceles triangle (a triangle having two equal sides) to its base the original triangle is divided into two similar or two symmetrical triangles. If the base of this perpendicular is moved to either side, then these two similar triangles become dissimilar or asymmetrical. The converse of this geometric proposition is equally true. The base of the nose as viewed from below is an isosceles (the two sides are equal) triangle. From the apex of this isosceles triangle the lower end of the cartilaginous septum, together with the columnar cartilages, forms a perpendicular to the base line and divides the triangular base of the nose into two symmetrical or two similar nares.

If the perpendicular or lower end of the cartilaginous septum is shifted or sprung to either side from out of its septal groove or normal midline position in the floor of the nose, then the two normally similar triangular nares or symmetrical nares are rendered dissimilar or asymmetrical.

The converse of this geometric proposition can be readily demonstrated by replacing or resetting the dislocated lower end of the cartilaginous septum back into the midplane into the normal septal groove in the floor of the nose, thereby reforming or reestablishing the two similar or two symmetrical nares. The truth of this geometric proposition and its converse as applied to the dislocation of the lower end of the cartilaginous septum and the nares permit the statement of the following principle.

PRINCIPLE [1]

When following a nasal traumatism in a previously normal nose in an adult, in a child or even in the new-born there results a definite asymmetry of the nares. This altered condition may be interpreted as

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Read before the Philadelphia Laryngological Society, Nov. 3, 1931.

1. In 1916, at the American Medical Association meeting in Detroit, the principles announced in this paper were brought out in a Scientific Exhibit on "Nasal Reconstructive Surgery." In 1930, at the Scientific Exhibit at the American Medical Association meeting in Detroit, this same principle was again emphasized, entitled, "Resetting of the Nasal Septum Versus the Submucous Resection."

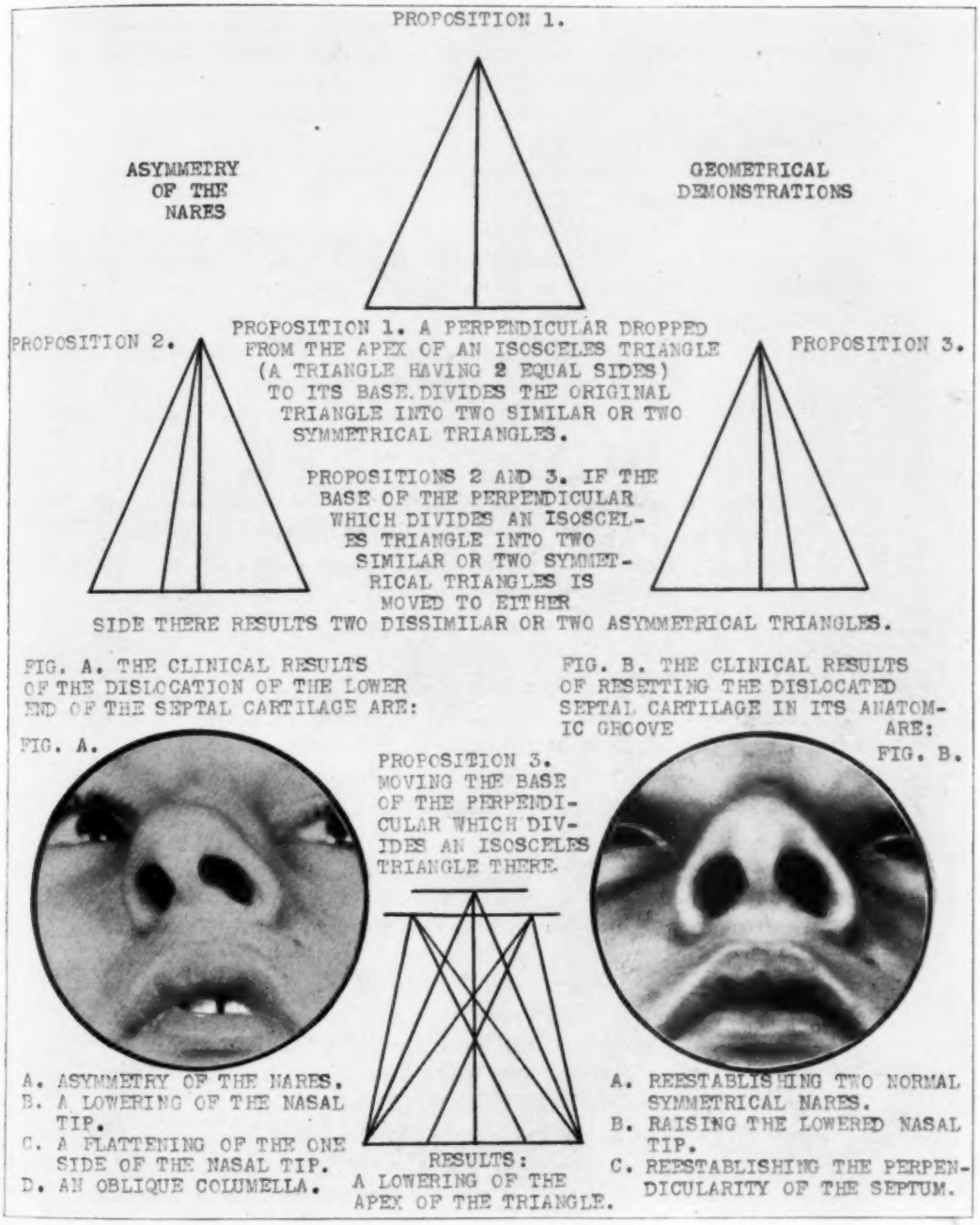
PROPOSITION 1.
ASYMMETRY OF THE NARES
GEOMETRICAL DEMONSTRATIONS
PROPOSITION 1. A PERPENDICULAR DROPPED FROM THE APEX OF AN ISOSCELES TRIANGLE (A TRIANGLE HAVING 2 EQUAL SIDES) TO ITS BASE, DIVIDES THE ORIGINAL TRIANGLE INTO TWO SIMILAR OR TWO SYMMETRICAL TRIANGLES.
PROPOSITION 2.
PROPOSITION 3.
PROPOSITIONS 2 AND 3. IF THE BASE OF THE PERPENDICULAR WHICH DIVIDES AN ISOSCELES TRIANGLE INTO TWO SIMILAR OR TWO SYMMETRICAL TRIANGLES IS MOVED TO EITHER SIDE THERE RESULTS TWO DISSIMILAR OR TWO ASYMMETRICAL TRIANGLES.
FIG. A. THE CLINICAL RESULTS OF THE DISLOCATION OF THE LOWER END OF THE SEPTAL CARTILAGE ARE:
FIG. B. THE CLINICAL RESULTS OF RESETTING THE DISLOCATED SEPTAL CARTILAGE IN ITS ANATOMIC GROOVE ARE:
FIG. A.
FIG. B.
PROPOSITION 3. MOVING THE BASE OF THE PERPENDICULAR WHICH DIVIDES AN ISOSCELES TRIANGLE THERE
A. ASYMMETRY OF THE NARES.
B. A LOWERING OF THE NASAL TIP.
C. A FLATTENING OF THE ONE SIDE OF THE NASAL TIP.
D. AN OBLIQUE COLUMELLA.
RESULTS: A LOWERING OF THE APEX OF THE TRIANGLE.
A. REESTABLISHING TWO NORMAL SYMMETRICAL NARES.
B. RAISING THE LOWERED NASAL TIP.
C. REESTABLISHING THE PERPENDICULARITY OF THE SEPTUM.

FIG. 1.—GEOMETRIC DRAWINGS OF ASYMMETRY OF THE NARES.

a positive diagnostic sign (or entity), establishing the anatomic displacement of the lower end of the cartilaginous nasal septum from the septal groove in the floor of the nose with or without other anatomic displacements of the bones and cartilages of the framework of the nose.

Other anatomic displacements of the nasal framework which may and usually do result from the dislocation of the lower end of the cartilaginous nasal septum are:

Corollary 1.—The normally perpendicular columella assumes an oblique position (figs. 1, 2 *A*, 6 *B* and 7 *B*).

Corollary 2.—The nasal tip is lowered, owing to the partial removal of the supporting end of the cartilaginous septum from underneath the lower lateral cartilages and from its supporting position back of the columella (figs. 1, 2 *A*, 6 *B* and 7 *B*).

Corollary 3.—The free lower end (border) of the septal cartilage covered by mucous membrane is seen lying partially across either nostril instead of being hidden posterior to the columella in the midplane (figs. 4 *A* and 5 *A*).

Corollary 4.—Higher up in the opposite nostril there is a rather sharp "angle of deflection (Metzenbaum)" of the septal cartilage, resulting in a partial or nearly total obstruction to this nostril (fig. 5 *A*).

Corollary 5.—Either side of the nose appears somewhat flattened from its tip to the outer attachment at its base like the one flattened nostril following the repair of a cleft palate harelip (figs. 1, 2 *A*, 6 and 7).

Corollary 6.—The dorsum of the nose at its tip is flattened and has a depression as a result of a partial separation of the two lateral cartilages and the cartilages in the columella (fig. 4 *B*).

Corollary 7.—The lower cartilaginous half of the nose forms an angle "false nasal hump (Metzenbaum)" with the lower end of the nasal bones (fig. 8 *A*).

Especially in infants and in young children, nasal injuries of a lesser degree and nasal injuries of a greater degree are often left untreated because there are no commonly known diagnostic signs establishing the fact that the lower end of the septal cartilage has been dislocated from the groove in the floor of the nose. The swelling and contusion of the surrounding soft tissues usually obscure the entire field. There is also a common lack of knowledge that the lower end of the septal cartilage may be dislocated from the groove in the floor of the nose in the newborn, in the infant and in young children (Metzenbaum).

Roentgen examination of the nose is almost without value in young children when the injury involves only the cartilaginous septum, and it is of real value only in establishing the diagnosis of the bony parts of the nose in cases in which there is considerable bony separation. Physical examination soon after injury may fail to reveal the lesser

fractures and dislocations of the framework of the nose, owing to the great amount of swelling and hematoma of the soft tissues. The displacement of the lower end of the cartilaginous nasal septum is among the most frequent injuries to the nose from infancy to about the age of 10 years (Metzenbaum), and almost all other injuries to the nose, excepting the injuries involving the nasal bones per se, may and usually will include the displacement of the lower end of the cartilaginous septum.

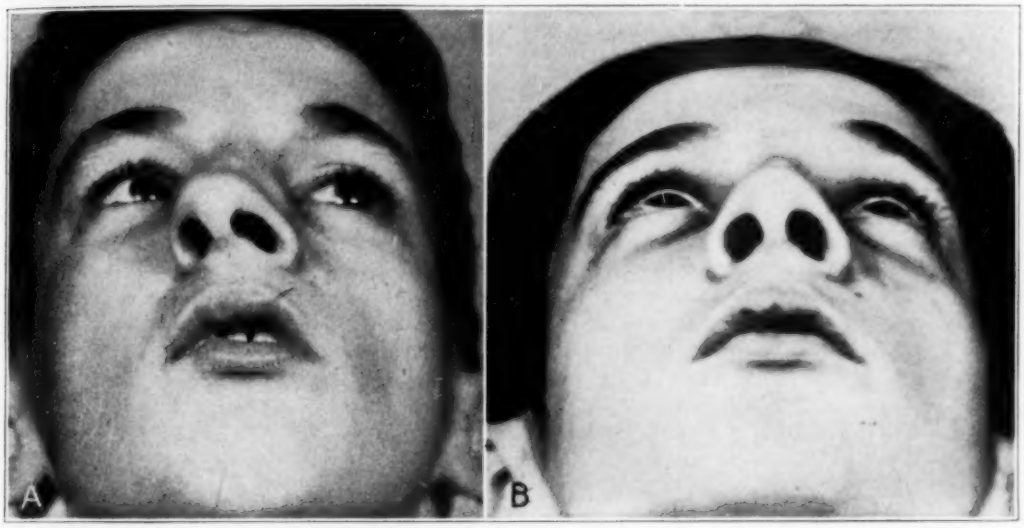

Fig. 2.—*A*, results following the dislocation of the lower end of the septal cartilage from the groove in the vomer and from the "maxillary groove (Metzenbaum)" are: (1) asymmetry of the nares; (2) an oblique columella; (3) the dislocated lower end of the septal cartilage in one (right) naris is not seen in this view; (4) the tip of the nose is lowered and closer to the upper lip, owing to the partial removal of the support of the lower end of the septal cartilage from underneath the lower lateral cartilages; (5) the "angle of deflection (Metzenbaum)" of the dislocated septal cartilage somewhat higher in the left (opposite) naris causes a partial occlusion of the left (opposite) naris; (6) the left side of the nose is flattened from its tip to the outer attachment like the flattened nostril following the repair of a cleft palate harelip. *B*, results following the resetting of the dislocated posterior border of the septal cartilage into its anatomic groove in the floor of the nose reestablishes: (1) two normal symmetrical nares; (2) a perpendicular columella, (3) the free lower end of the cartilage is replaced back of the columella; (4) the lowered nasal tip is raised, reestablishing the normal perpendicularity of the lower end of the cartilaginous septum, a factor on which normal nasal function depends (Metzenbaum); (5) the partially obstructing "angle of deflection (Metzenbaum)" of the septum in the left naris is brought into the straight midplane position, thereby rendering the left naris patent; (6) the total result of the resetting of the dislocated septal cartilage into its normal anatomic position reestablishes normal nasal function.

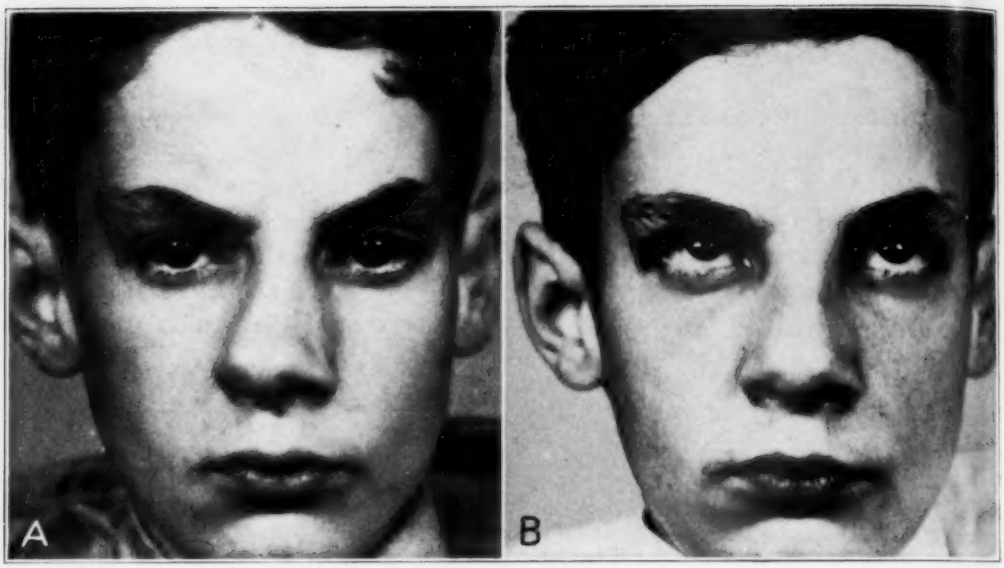

Fig. 3.—*A*, injury to the nose in early childhood. The entire cartilaginous septum was dislocated from the shallow anatomic groove in the vomer and from the shallow maxillary groove and sharply deflected toward the right, resulting in a partial obstruction to both nares. *B*, the resetting of the dislocated septum back into the shallow anatomic groove in the vomer and back into the shallow anatomic maxillary groove in the midplane position in the floor of the nose reestablished normal nasal function, namely, the ingress and egress of air.

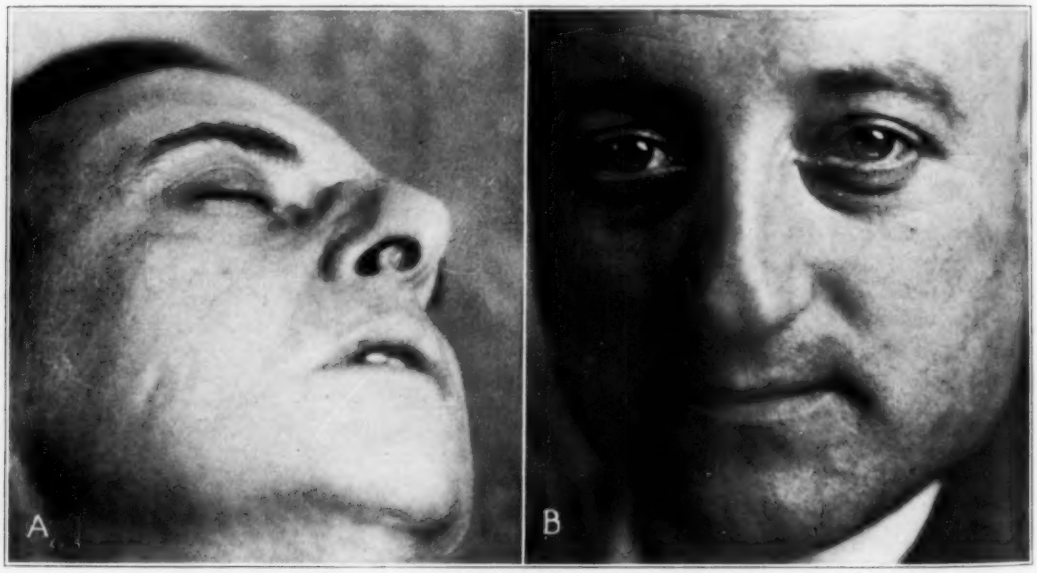

Fig. 4.—*A*, nasal injury in early childhood resulting in a dislocation of the lower end of the septal cartilage with (1) a dislocation of the lower end of the right columnar cartilage and (2) a separation of both cartilages in the columella. *B*, nasal injury in early childhood with a dislocation of the lower end of the septal cartilage resulting in (1) a lowering of the nasal tip and (2) a definite depression at the tip of the nose due to a wide separation of the lower lateral cartilages.

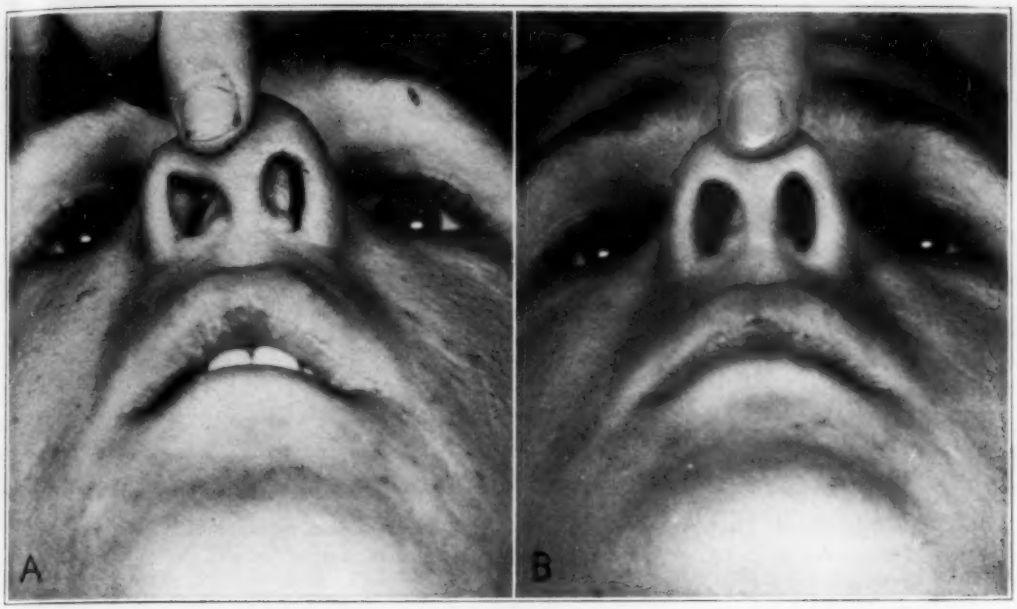

Fig. 5.—*A*, injury to the nose in early childhood: (1) dislocation of the lower end of the septal cartilage; (2) the free lower end of the cartilage is seen lying partially across the right nostril; (3) higher up in the left nostril is seen the sharp "angle of deflection (Metzenbaum)." *B*, the reset cartilage shows about 80 per cent anatomic restoration: (1) symmetrical nares; (2) perpendicular septum; (3) a raising of the tip of the nose; (4) restoration of function.

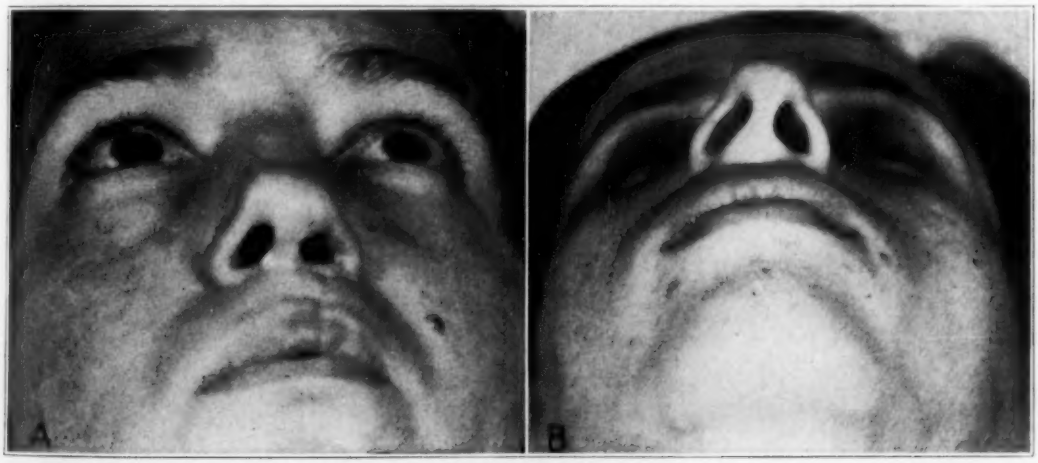

Fig. 6.—*A*, patient with a repaired cleft palate harelip due to a failure to reset the lower border of the septum in its normal anatomic position in the midplane of the floor of the nose. The resulting anatomic displacements of the nasal framework are similar to those resulting in a case of dislocation of the lower end of the septum, namely: (1) asymmetry of the nares; (2) an oblique columella; (3) the tip of the nose lowered; (4) a flattened nasal tip on one side to its outer attachment at the base. *B*, patient with a dislocation of the lower end of the cartilaginous septum, showing anatomic displacement similar to the repaired cleft palate harelip, namely: 1, 2, 3 and 4 in *A*.

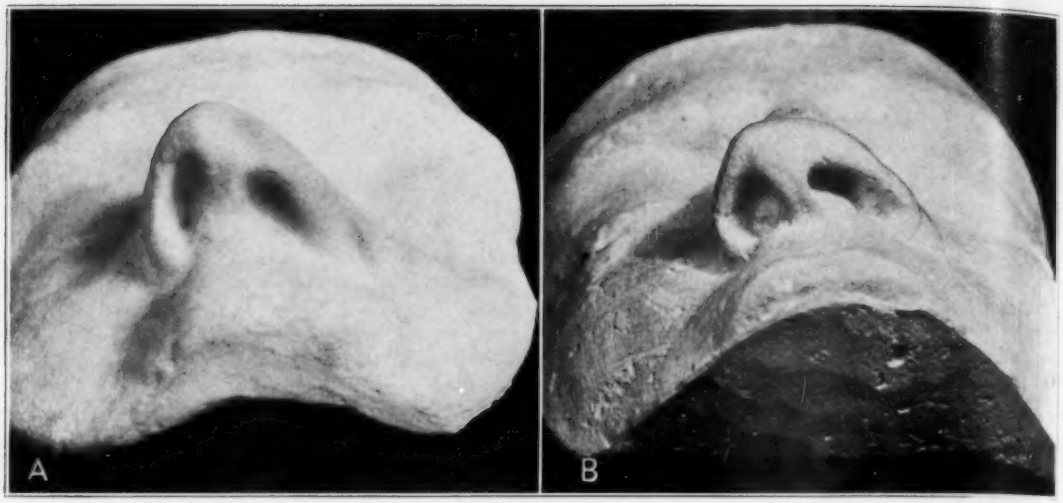

Fig. 7.—*A*, plaster cast of the nose of a patient with a repaired cleft palate harelip due to a failure to reset the lower border of the septum in its normal anatomic position in the midplane of the floor of the nose. The resulting anatomic displacements of the nasal framework are similar to those resulting in a case of dislocation of the lower end of the septum, namely: (1) asymmetry of the nares; (2) an oblique columella; (3) the tip of the nose lowered; (4) flattened nasal tip on one side to its outer attachment at the base. *B*, plaster cast (from the same case as photographed in fig. 2 *A*) with a dislocation of the lower end of the cartilaginous septum, showing similar anatomic displacements to the cast of the repaired cleft palate harelip, namely: 1, 2, 3 and 4 in *A*.

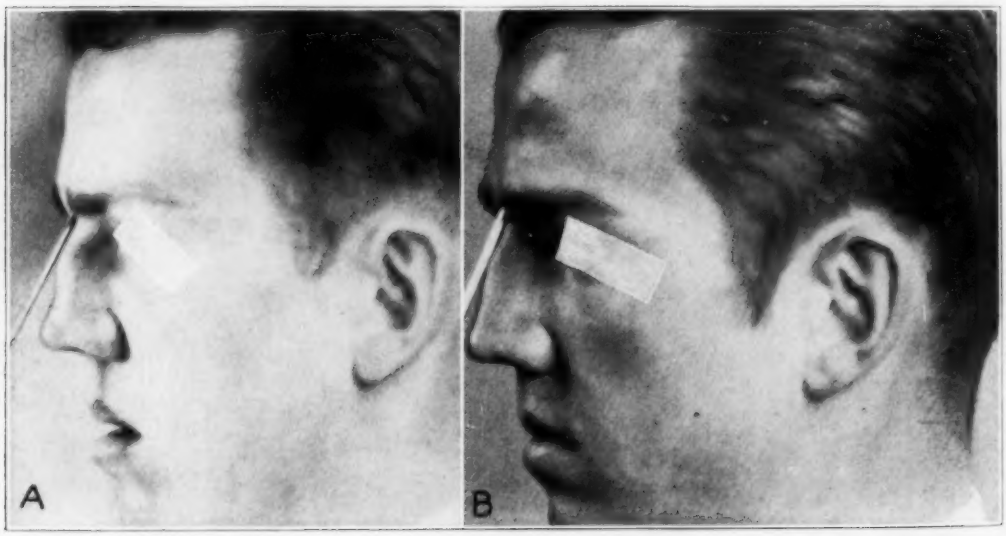

Fig. 8.—*A*, injury sustained in early childhood, resulting in a dislocation of the lower border of the cartilaginous septum from the entire length of the groove in the vomer and the maxillary groove resulting in the formation of a "false nasal hump (Metzenbaum)" of the septal cartilage and the normal nasal bones. *B*, the dislocated septal cartilage was reset in the septal groove in the floor of the nose, reestablishing the normal perpendicularity of the cartilaginous septum, resulting in the restoration of normal nasal function (Metzenbaum).

The dislocation of the lower end of the septal cartilage may result in a partial or a total obstruction to one or to both nares, which obstruction interferes with the normal nasal function, namely, the ingress and egress of air (through the nares). When this septal obstruction occurs in the infant or the young child, it may be and usually is a contributing factor to the asymmetrical and the inharmonious development of the nose and possibly to the upper jaw and the upper part of the face.

The recognition of these anatomic displacements soon after the time of injury and their simple reduction will usually:

(*a*) Reestablish normal nasal function.

(*b*) Aid in the normal development of the child's nose (jaws and face).

(*c*) Prevent the short underdeveloped columella.

(*d*) Prevent deformities of the nasal tip.

(*e*) Insure normal development of the nasal tip.

(*f*) Obviate the usual operation for the submucous resection of the cartilaginous septum "after nasal development has taken place from the sixteenth to the eighteenth year."

In cases in which sufficient time has elapsed for the exudate to organize into a callus and fill in the "maxillary groove (Metzenbaum)" and the groove in the vomer and incorporate the base of the dislocated cartilage firmly to the sides of the vomer and the maxillary spine, then in order to reset the dislocated cartilaginous septum, it becomes necessary to follow a definite surgical procedure for the resetting of the dislocated lower end of the septum in children. For this type of case I have devised an operation known as the "resetting of the old nonreduced lower end of the dislocated cartilaginous septum versus the submucous resection." [2] I have performed this operation during a period of fifteen years:

(*a*) None of the septal cartilage has been removed.

(*b*) In about from 80 to 85 per cent of the patients operated on the reset lower end of the dislocated septal cartilage remained in the anatomic or near anatomic position.

(*c*) Good nasal function was reestablished.

(*d*) There was slight or no deformity of the nasal tip.

(*e*) There was improved or normal development of the nose (the jaws and the face) thereafter.

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2. Metzenbaum, Myron: Replacement of the Lower End of the Dislocated Septal Cartilage Versus Submucous Resection of the Dislocated End of the Septal Cartilage, Arch. Otolaryng. **9**:282 (March) 1929.

MAXILLARY SINUSITIS

PRACTICAL CLINICAL SUGGESTIONS ON DIAGNOSIS AND TREATMENT

WILLIAM WESLEY CARTER, M.D.
NEW YORK

The marvelous development of the various radical operations on the paranasal sinuses—and one must admit that they have well nigh reached the point of perfection from a mechanical point of view—has inspired confidence in them, especially among the younger rhinologists, that is unwarranted by the ultimate clinical results.

An operation may be a triumph of perfection in the eyes of the mechanical genius who devised it, or of the skilful operator who faithfully followed his directions in every particular; yet the same operation may be pronounced a colossal failure by the patient, who during the remainder of his life may have to contend with a pus-secreting, scabbing area in his nose, a constant menace to his health and happiness.

There are occasions when radical operations on the sinuses not only are indicated, but are absolutely necessary; a decision, however, to operate, save in an emergency, should not be made until the case has been thoroughly studied and the situation discussed in consultation with at least one other physician of experience in this field.

Enthusiasm over radical surgical interventions on the sinuses has waned decidedly within the last three or four years, and the indications for this treatment are now scanned more closely than ever. The result is that fewer radical operations on the sinuses are now done, especially by those who base their decisions on the knowledge gained during many years of clinical experience, for they know that these radical operations, it matters not how skilfully they are performed, are sometimes followed by results that are everything but satisfactory to the patient. Many now approach the subject of radical procedures on the paranasal sinuses in a spirit of contrition and with the fear of God in their hearts. This is not because of timidity or lack of confidence in the mechanical efficiency of a radical operation, but rather because of solicitude concerning the future comfort of the patient.

It is a great mistake for a surgeon to pronounce an operation a success simply because he has succeeded in solving a mechanical problem and finds when he is through operating that the physical results of his work are satisfactory; time is the final arbiter in this matter.

"Experience is the best teacher." This terse, well established verdict has been accepted through the ages by those who have lived and have evaluated the phenomena of life.

In the acquisition of experience, the element of time is essential. This applies especially to the problems that confront the rhinologist, for he must anticipate the results of his labors, which in many instances cannot be accurately evaluated until some time after the operation, for the possibility of sequelae must always be taken into consideration.

The maintenance of the paranasal sinuses in a state of health is dependent on free aeration and drainage. A sinus is healthy when its ciliary epithelium is performing its function, namely, that of keeping the flow of the secretions directed toward the ostium, which, it is unnecessary for me to say, must be open and in free communication with the nasal cavity. Under these conditions the sinuses resist infection and do not become clogged with secretions of any kind.

Paralysis of the cilia quickly follows irritation. The most common cause is a bad cold. If this inhibition of their action is prolonged, the cilia are destroyed, permanent pathologic changes occur in the underlying tissue, and the condition passes into the chronic stage. If this phase persists long enough for the cilia over the entire sinus wall to be destroyed, there is no treatment that will restore the sinus to a normal state, for ciliated epithelium is never regenerated following its complete destruction by disease, by operative measures or in any other way. Ciliated epithelium is a differentiated, highly specialized tissue, and its function is chiefly mechanical. For its development, its physical qualities and its function it is absolutely dependent on the primitive germ cell. Therefore, if it is eradicated, it cannot be replaced, at least by regeneration from underlying basal tissue, any more than can any other organ in the body once it has been destroyed or removed. Full thickness of skin is never regenerated over a denuded area, so why should ciliated mucous membrane act otherwise? It is conceivable that, under favorable conditions, ciliated epithelium might spread from an adjacent normal area and cover a limited surface that had been deprived of its normal covering. I have never noted such a phenomenon, however, and I believe that if this did happen it would be of no clinical value, because the area covered would be inconsiderable and of doubtful functional utility because of the underlying scar tissue.

It follows, therefore, that in any operation in which the ciliary lining is destroyed the sinus is left in an abnormal condition, for its inherent power to cleanse itself has been lost.

In all radical procedures on the sinuses, painstaking efforts should be made to save all healthy tissue that may be found in the area on which operation is performed.

In the treatment of acute sinusitis the most that one can expect to accomplish is to establish aeration and free drainage and to build up the patient's general resistance to infection; in other words to assist nature in her efforts to maintain a healthy sinus. This should be done, if possible, by palliative and not by operative measures. It is known that an operation on an inflamed, acutely infected mucous membrane in the nose or throat is a hazardous procedure; the danger of inciting septicemia or meningitis is great; no surgeon should accept the responsibility of operating during the fulminating stage of an infection, save in an extreme emergency, and even then his activities should be confined to the establishment of aeration and drainage.

I may say here that the management of sinusitis during its acute and subacute stages is one of the most important problems with which the rhinologist has to deal, for at this critical period he may, by judicious treatment, forestall the development of chronic sinusitis. This is true especially in the case of children. A bad cold that lasts over two weeks is always a case of sinusitis and should be treated as such. Much suffering and impairment of health in later life might be avoided by adopting such a course.

It is seldom necessary to operate on a child's sinus, for it can be successfully treated by irrigations though the ostium. This opening is relatively larger in the child than it is in the adult. Furthermore, the floor of the child's maxillary sinus is on a higher level than the floor of the nasal cavity and remains so until the twelfth year, when it dips down below the floor of the nasal cavity and gradually assumes its normal adult position, which is considerably below the floor of the nose. These anatomic differences favor both drainage and the treatment of the maxillary sinus of the child by irrigation through the ostium.

The etiology, diagnosis, prognosis and treatment of chronic antral disease is so intimately associated with and is so dependent on the correct determination of the character of the contents of the cavity and the condition of its walls that treatment, attempted before acquiring this knowledge, would be practically on an empiric basis.

In making a study of disease of the antrums one should apply all of the diagnostic aids, for each of these not only contributes its own special information, but amplifies the knowledge gained from the other tests. No single test is sufficient in itself to give one comprehensive knowledge of the situation.

The chief aids to the diagnosis of sinus disease are transillumination, roentgen examination, washing out of the sinus either through its ostium or by puncture through the inferior meatus and the antroscope.

It often happens, even after all these tests have been made, that the exact nature of the contents of the sinus and the condition of its walls

have not been revealed, and surprises sometimes await one when the interior of the antrum is inspected directly through an operative opening in the anterior wall.

The methods of applying the various diagnostic tests are familiar to all and will not be described, but I should like to record my own reaction to some of the modifications in the application of these tests.

In transilluminating a maxillary sinus, I often get a good idea of the extent of its involvement and the relative translucence of the two sides by placing the light over the floor of the orbit and in various positions over the cheek and alveolar process and then evaluating the transmission of light through the roof of the open mouth.

The value of roentgen examination is unquestioned, but it cannot be relied on to the exclusion of other tests, as all rhinologists know from clinical experience. Standardized positions of the head in making films of the sinuses, such as those devised and utilized by Dr. F. M. Law of New York are as indispensable as are the expert interpretations of the films and consultations with the roentgenologist.

The value of radiopaque substances, such as iodized poppy seed oil 40 per cent, injected into the sinus before the roentgen examination is made is questioned by many clinicians. Personally I feel that nothing is to be gained by the use of such substances, for there are several sources of error, and the results may be misleading. If the sinus has not been completely cleared of its secretions, especially inspissated pus, before the radiopaque substance is injected, a false impression of the condition of the sinus walls will be conveyed. If, perchance, a tumor springing from the sinus wall is completely surrounded by the opaque material, no intimation of its presence will be given by the x-rays. Under such circumstances the entire sinus will appear to be filled with the radiopaque substance in the plate, and one may conclude that the sinus is normal and may not even suspect the presence of a tumor.

Radiopaque substances merely give a profile outline of a plane of the sinus cavity corresponding to its widest and longest diameters from a certain point of view. A radiopaque substance does not give reliable information concerning either the contents of the sinus or the condition of its walls.

In probably 75 per cent of patients the antrum may be satisfactorily washed out through the ostium, but when there is inspissated pus thickly spread over its entire floor I question whether this can be thoroughly done through a single opening which is used at the same time as both the inlet and outlet for the solution.

In adult patients I prefer to wash out the antrum through a cannula, inserted by way of the inferior fossa, using the ostium as a counter-opening for the exit of the solution.

If the washings are collected in a black pus basin, much may be learned of the condition of the interior of the cavity.

In my opinion, a patient suffering from chronic antral disease should be given the benefit of conservative treatment, that is, lavage, either through the ostium or through the inferior fossa twice a week for a considerable period, if necessary. No operation should be considered until one is convinced that the conservative method is not producing the desired result.

Of course, if the antrum contains adventitious tissue of any kind, such as granulation tissue or new growths, or if the bony walls have become necrotic, the indications are clear that nothing short of a radical operation will do the patient any good. The Caldwell-Luc operation is the best procedure. This operation provides the best permanent drainage for both the antrum and the ethmoid area. In cases requiring radical operation on the antrum, the ethmoid labyrinth is practically always involved, and, unless this area is also drained, the infection is prolonged indefinitely.

At best, the radical operation, I must frankly admit, is a choice of evils, and the patient must be made to realize that for at least a considerable period he must suffer the discomfort of a mucopurulent discharge and the occasional expulsion of scabs from the nose.

Infected teeth must always be kept in mind as an etiologic factor in maxillary sinusitis. Should this source be suspected, a competent dentist should be consulted at once. If a sinus is found to be infected from an apical abscess, the tooth should be removed. In my experience, when an abscess has progressed far enough to cause maxillary sinusitis, it cannot be successfully treated by conservative methods; time, suffering and money will be saved by prompt removal of the tooth.

Dental sinusitis is due to infection at the apex of a tooth that protrudes into the floor of the antrum. Consequently when the tooth is extracted there is established an opening into the mouth through the alveolar process. Because of crooked or hooked roots or other anatomic irregularities, a portion of the floor of the sinus may be removed during the extraction, thus making the opening into the mouth large. This unwelcome occurrence may be no fault of the dentist; in many instances it cannot be avoided.

Following the extraction of the tooth, under no circumstances should irrigation of the antrum be practiced through this opening. The infection would be spread by such a procedure, and there would be no opportunity for the opening to close. One's efforts, under these circumstances, should be confined to keeping the mouth clean and the wound free from particles of food.

If irrigation of the sinus is found to be necessary, it should be done through the ostium or through a cannula by way of the inferior fossa.

Fortunately, nature usually takes care of these cases as soon as the cause of the infection has been removed, provided that the irritation has not continued long enough for extensive pathologic changes to have occurred in the lining membrane of the sinus.

Meddlesome interference with dental sinusitis by the dentist or the surgeon accounts for many cases of serious antral disease in which there is a persistent discharge of pus into the mouth through the alveolar opening. Sometimes it is almost impossible to close these openings.

In these days when focal infections occupy a reserved seat in the minds of both the members of the medical profession and the laity, one would not think of neglecting this possibility in discussing the infections of the sinuses.

There is a tendency to attribute to the sinuses many aches and pains that cannot otherwise be accounted for. Apparently this is due, in part at least, to the fact that these cavities are not open to direct inspection, and that even roentgen examination does not reveal some pathologic conditions. Hence the mysteries of their hidden recesses permit the imagination to run riot. Consequently many ineffectual operations are performed and residual conditions are added to the original discomforts of the patient.

Only a month ago I saw a woman whose right ethmoidal labyrinth had been completely exenterated. She said that for several years she had suffered at times with severe pain in the right side of her head, and that she often had vague sensations of discomfort, but seldom actual pain, in some of her joints. It was for these symptoms that she sought relief.

This woman assured me that prior to the following experience, she had never had any trouble with her nose. The physician whom she consulted said that she had a cyst in the right middle turbinate and attributed her troubles to this. He operated, but the patient was not benefited, and now, though only 40 years of age, she must go through the remainder of her life suffering from the discomforts I have mentioned that so frequently follow radical operations on the ethmoid sinuses. The regrets in this instance are intensified by the fact that her nose was perfectly normal before the operation.

Empiricism in surgery is seldom warranted, save in emergencies. Never should an operation be performed without mature deliberation and seldom without consultation with an experienced confrère, especially when there is only a chance of relieving the patient and a certainty of leaving her maimed, as in the case I have just cited.

It is my belief, based on clinical experience, that infections seldom emanate from foci located in the paranasal sinuses. I attribute this to the fact that aeration and drainage are usually adequate to insure against the retention of infectious material under pressure and also to what

appears to be a clinical fact, namely, that absorption into the general circulation of toxic material by way of the lining mucous membrane of the sinuses occurs with neither the same constancy nor the same rapidity as in other parts of the body. This peculiarity is emphasized in the chronic cases, owing to the fibrotic changes that have occurred in the mucous membrane lining these cavities.

It is true that many cases of asthma are due to sinus infection. In many of these the condition may be benefitted or cured by operation. On the other hand there are many sufferers who have not been helped in the least by operation on the sinuses. It must be remembered that asthma is so closely related to allergy and to biochemical dysfunction that these conditions must always be eliminated by proper tests before operation is advised; otherwise the patient will not be benefited by any surgical procedure on the sinuses.

In any case of asthma, even when the sinuses are seriously involved and must be operated on anyway, one should be guarded in prognosis so far as relief of the asthma is concerned. This is true especially when the patient has suffered with asthma for many years and the perverted nerve paths have become well established.

In my brief presentation of this important subject, I have purposely avoided making a formal scientific classification of the various manifestations of sinus disease, for I do not wish to becloud the practical significance of my comments by the introduction of material having chiefly academic interest.

In summarizing, I wish to emphasize the following points which have been evolved and evaluated from my personal clinical experience:

1. A sinus cannot be healthy without free aeration and drainage.

2. Healthy cilia indicate a healthy sinus. If ciliated epithelium is completely destroyed, it is never regenerated. In all operations, therefore, one should save as much of the lining membrane as possible.

3. Iodized poppy seed oil 40 per cent is not to be relied on in determining either the contents of the sinus or the condition of its walls.

4. Adventitious tissue in the antrum is, as a rule, an indication for a radical operation. This should be thorough, the Caldwell-Luc procedure being best suited for most of these cases.

5. Maxillary sinusitis, primarily of dental origin, is unusual. Most of these cases are the result of meddlesome interference with infected root cavities after extractions.

6. Focal infections from the sinuses are probably of rare occurrence; they are difficult to trace, and the conclusions are often clouded by uncertainty.

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DYNAMICS OF NASAL RESPIRATION

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In seeking data on the physiology of nasal obstruction it is noteworthy that most of the prominent textbooks on the nose and throat fail to give separate consideration to that subject per se, and for its interpretation the reader is obliged to form his own deductions about a condition that should convey a more definite picture than is offered at the present time. On that account, the term "nasal obstruction," like "rheumatism," has become a frequent but loose expression to denote mild or severe degrees of nasal closure, and in a number of instances a mere subjective state not supported by clinical observation. By creating an issue as to the importance of an intelligent approach to this important problem, much may be done to arrive at more definite indications for nasal surgical treatment for this condition which in the present state of investigation cannot always stand the test of critical analysis.

One may ask oneself: Is one justified in correcting nasal or septal deformities when there is no evidence of functional impairment? Is one to think in terms of structure or of function? One not infrequently encounters cases of fossal closure in patients who do not complain of nasal respiratory embarrassment, and, vice versa, others with marked patency are just as likely to complain of obstruction. Something else is required to confirm or negate the complaints of the patient or the findings of the rhinologist. To diagnose a case as hypertension without a manometric record is folly. Might not a pressure or volume record of nasal respiration be of some value?

European colleagues have in recent years manifested a serious interest in manometry as applied to nasal function. In Denker and Kahler's "Handbuch der Hals-Nasen-Ohren-Heilkunde" [1] one reads of Zwaardemaker's physiologic experiments along this line, but one does not find any collaboration or application of these experiments by any of the clinical contributors to this work.[2] For his experiments Zwaardemaker employed a special apparatus of his own for measuring

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1. Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohren-Heilkunde, Berlin, Julius Springer, 1925.

2. Zwaardemaker: Physiologie der Nase und ihrer Nebenhöhlen, in Denker and Kahler (footnote 1, vol. 1, p. 439).

the volume of air which he called an "aerodromometer." He also carried out pressure tests with a water manometer.

Undritz,[3] a rhinologist of Leningrad, more recently sensed the need of applying such experiments as Zwaardemaker's to his clinical work, and with the assistance of physicists at the university of that city he constructed what he called a "rhino-anemometer" for measuring objectively the degree of capacity of the nasal passages, or the volume of air expired in a unit of time. He connects one of the nostrils with the water manometer for pressure and the other with his volume recording rhino-anemometer. The subject is then asked to perform a single but prolonged expiration at a constant pressure of 10 mm. of water, and the volume of air expired is recorded after the person has expended his expiratory force. Undritz has extended the uses of this device not only to diseases of the nose but also to diseases of the upper organs of respiration. In this respect, its methods and uses are not unlike those employed by Frost in his studies on cardiorespiratory function, except that the latter worker uses the buccal route for expiration and instructs his patients to maintain twenty-seven times as much pressure (20 mm. of mercury) as Undritz.

Worms and Bolotte[4] have made further contributions to the clinical side of nasal function and have embodied their observations in a simple and well written book titled "L'insuffisance respiratoire nasale," in which they illustrate numerous devices designed to assist the rhinologist in properly interpreting nasal obstruction. From a study of numerous subjects with normal and obstructed noses, respectively, they confidently concluded that volumetric studies in relation to nasal obstruction are unreliable. In partial nasal obstruction they found the volume of air passing through the nose per unit of time often to be even greater than one would expect in a normal nose. In marked nasal obstruction, however, there was a constant diminution in volume of air per unit of time.

In my initial studies of nasal respiratory function I have, therefore, for the present, avoided the consideration of volumetric studies of the nose, because: (1) the spirometers as constructed at the present time require a conscious effort on the part of the subject to produce any appreciable readings and no volume recorder to my knowledge is sensitive enough to tell how much air a person can inhale or exhale in a normal natural breath of negligible pressure, and (2) pressure studies in my preliminary investigations have led to certain conclusions which appear satisfactory.

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3. Undritz, W.: The Rhino-anemometer, Acta oto-laryng. **14**:513, 1930.

4. Worms and Bolotte: L'insuffisance respiratoire nasale, Paris, A. Maloine et fils, 1928.

The type of manometer was, of course, an important consideration. What pressure manometer would record quiet inspiration or expiration? What type would record forced nasal inspiration or expiration? The aneroid manometer was found to be too insensitive for fine readings. The familiar U gage was found to be the simplest and least expensive and, moreover, could be depended on for approximate readings. The fine micro-anemometer devised by Krell and manufactured by the Cambridge Scientific Instrument Company would probably have been ideal for accurately recording low pressures. One can readily understand that in such states as coma the readings might be so low as to produce no appreciable fluctuations of the water manometer. The type of gage fluids was also an important problem. Two U gages were required, one for recording low pressures, the other for forced pressures. For the more sensitive gage, a fluid of low specific gravity was needed. Alcohol would fall into this class, but as its weight is not much less than that of water, the latter medium was selected. For forced nasal pressures water would also have been an excellent medium, but that would have entailed such an enormously high U tube that a heavier fluid was sought. Salts, such as magnesium sulphate or sodium carbonate, by their dissolution in water increase its specific gravity but not sufficiently to warrant their substitution. Besides, there is a sedimentation of the salts on the walls of the tubes after a little usage. After trials with these and other substances, I was obliged to employ the much heavier (13.5×) mercury for the forced pressure readings.

The tests were carried out in systematic fashion and were recorded as follows:

I. Forced inspiration (mercury manometer), in millimeters of mercury
   - (*a*) Right nasal
   - (*b*) Left nasal
   - (*c*) Binasal
   - (*d*) Mouth
     1. Postoral
     2. Suction
   - (*e*) Difference in pressures on both sides (in millimeters of mercury)

II. Forced expiration (mercury manometer), in millimeters of mercury
   - (*a*) Right nasal
   - (*b*) Left nasal
   - (*c*) Binasal
   - (*d*) Mouth
     1. Oral
     2. Buccal
   - (*e*) Difference in pressures on both sides (millimeters of mercury)

III. Natural inspiration (water manometer) in millimeters of water
  (*a*) Right tubal
  (*b*) Left tubal
  (*c*) Postoral
  (*d*) Difference in pressures on both sides (in millimeters of water)

IV. Natural expiration (water manometer), in millimeters of water
  (*a*) Right tubal
  (*b*) Left tubal
  (*c*) Postoral
  (*d*) Difference in pressures on both sides (in millimeters of water)

V. Breaths per minute with natural breathing

VI. Subjective complaint

VII. Clinical findings

VIII. Difference in millimeters of mercury of forced right nasal inspiration and expiration
  Difference in millimeters of mercury of forced left nasal inspiration and expiration

IX. Difference in millimeters of water of natural right inspiration and expiration
  Difference in millimeters of water of natural left inspiration and expiration

In testing the patency of the left nasal fossa by the forced method (forced left nasal inspiration), as shown in figure 1 *A*, the nasal tip is tightly inserted into the left nostril while the right nostril is firmly occluded with the finger. The patient is then instructed to inhale with full force. The initial readings will be found to be valueless, because it takes a little time to make the subject understand what is required of him. With each attempt the result is found to be higher than the preceding one, and after several such attempts an average maximum will be reached. The patient is further warned not to inhale until he has had a satisfactory exhalation. He is also told not to make any efforts if there is any fluctuation in the mercury column. Three or four optimal readings are required to arrive at an approximate mean. For example, the forced inspiratory pressures on one side may read 40, 60, 50 and 50 mm. of mercury, respectively. Obviously, 50 mm. of mercury would be an average. The tip is next inserted into the left nostril, and the right one is occluded with the finger; the test is then repeated. The binasal tip is then inserted, as shown in figure 1 *B*.

In a normal case both sides will register equal values. The binasal reading will approximate the uninasal pressure. As will be seen in the case reports, a diminution in forced pressure on one side as compared with that on the other side points to homolateral nasal obstruction. It should again be emphasized that there should be no fluctuation of the mercury column as the patient begins the respiratory test; otherwise, the readings will reach erroneous heights. The rise in the mercury column should be steady.

The forced inspiration by mouth may yield deceptive readings unless it is carried out properly. One must bear in mind that the suction (fig. 1 *C*) pressure is much higher than purely oral inspiration, in which the tube is inserted 5 or 6 cm. into the oral cavity to abolish the element of suction. This latter pressure I have termed postoral for want of a better term. Oral pressure readings are used for control purposes. It is to be assumed that if the nose is markedly obstructed the mouth will not share in such a disturbance. Then again, the nose may be equally obstructed on both sides and yet the resultant figure will be meaningless. The pressure values in different persons vary, and in the same persons may vary at different times, and one may determine whether these variations are contributed by the nose or by the lungs as measured through the mouth pressures. In normal cases one may expect the postoral pressure

Fig. 1.—*A*, forced left nasal inspiration; *B*, binasal inspiration (forced); *C*, oral suction; *D*, left tubal pressure test (natural method).

to equal or be less than the nasal pressures. Only when that pressure is markedly greater than the nasal pressure has one reason for concern. In most instances the tests with the mouth may be dispensed with, as the nasal readings combined with clinical observations are fairly satisfactory. The subject must also be properly instructed on expiration through the mouth. The buccal expiration represents the increased effort induced by the muscles of the cheek as would be exerted with wind instruments and is higher than the true oral pressure, which is carried out by telling the patient to blow into the tube with the cheeks together in the same way as if he were blowing onto a hot beverage.

For forced nasal expiration the patient is instructed to inhale deeply before exhaling. He should also stop immediately if he feels an uncomfortable sensation in his ears. In my experiments I was fortunate in not receiving such complaints.

Natural inspiration signifies free natural breathing in a state of repose, or the calmest breathing a person with a nasal obstruction can carry out under the circumstances, and for this test the conscious element must be reduced to a minimum. In my initial tests I was not quite so careful to impress the importance of absolute calmness on the patient, and so while the readings did show quantitative differences in pressures on both sides, higher values were registered because the conscious element was not sufficiently reduced. The tip is tightly inserted in either nostril and the other nostril is left entirely free to carry on uninterrupted respirations (fig. 1 *D*). The patient is told to forget that there is a tube in one nostril, to read a paper or to look out of the window, to breathe as easily and as slowly as possible, and not to look at the water manometer. In this test, the initial readings will be found faulty, as the patient is at first quite conscious of the experiment. After about thirty seconds, the pressures will have adjusted themselves.

The natural postoral respiration method with the oral tube inserted 5 or 6 cm. into the oral cavity is recommended quite favorably by Worms and Bolotte,[4] who termed it the posterior rhinomanometry of Beyne. The mouth cannula is inserted with the lips tightly closed. The patient is allowed to breathe normally with the nose unobstructed and without conscious effort as in a state of repose, and the amplitude of oscillations is noted after a few moments. According to these observers, a negative inspiratory pressure of more than 15 or 20 mm. of water points to some nasal obstruction. My experiences with this method, as will be described later, are quite different. Only when both nostrils are closed can one expect increased oral pressures, and a complete unilateral nasal obstruction does not appreciably affect these oral pressures.[5]

REPORT OF CASES

For the sake of ready reference, I have given the names of the subjects for these tests.

CASE 1 (Myself).—Records of tests on myself as a normal control and two other sets of readings showing the effects of moderately and of tightly occluding one nostril artificially were made. By "moderate" occlusion I mean fairly tight plugging of one nostril with cotton. No matter how tightly I tried to plug the nostril with cotton, I was just barely able to reduce the forced pressures, and the significant changes were confined to the natural respiratory readings. By "tight" occlusion I mean firmly bridging the opening of the nasal tip (cannula) with a piece of adhesive plaster. It is interesting to observe that certain typical changes occur which coincide favorably with what I found in cases of marked clinical nasal obstruction.

5. One should always be careful to state pressures in terms of mercury or of water.

CASE 2 (Prinz).—Clinical right nasal obstruction with normal pressures (fig. 2). This patient did not complain of obstruction to nasal breathing, but was submitted to pressure tests for two reasons: (1) because of closure of the right nasal fossa due to a large inferior turbinate filling this space and (2) because alternate closure of the nostrils elicited easier breathing on the obstructed right side as compared to the left, which was roomy and showed somewhat atrophic changes in its mucous lining.

CASE 3 (Shaughnessy).—A case of typical "cold" with acute nasal obstruction on one side and an associated catarrhal sinusitis (fig. 3). The patient caught an "acute cold" with a discharge from the nose and "watery eyes" on the day previous to examination; she also had difficulty in breathing, especially through the left nostril, and pain over the left frontal sinus. There were marked congestion and edema of the mucous membranes, especially over the left turbinates. Left-sided obstruction was almost complete.

Two sets of records were taken in this case, before and after the application of a cocaine-epinephrine solution, respectively.

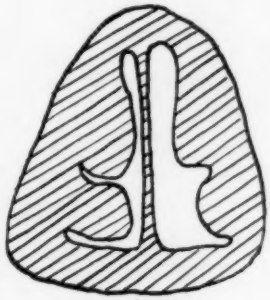


Figure 2

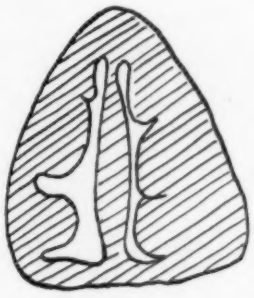


Figure 3

Fig. 2 (Prinz).—Pseudo-obstruction. Clinical right nasal obstruction; no subjective complaints; pressure tests normal.

Fig. 3 (Shaughnessy).—Acute left nasal obstruction, infectious. Objective obstruction and subjective obstruction; pressure tests confirmatory. Transitory condition, relieved by cocaine-epinephrine.

CASE 4 (Harrison).—Marked right septal deviation with right nasal obstruction (fig. 4). The patient had had frequent attacks of difficulty in nasal breathing, which seemed to alternate from one side to the other, although at the time of examination he was not certain which side was occluded until he was asked to close one nostril at a time. He attributed this condition to a kick in the nose while playing football many years previously. During the last three years the condition had become worse, due not to any additional blow, but to an aggravation of the condition with the passing of time.

The anterior septal deviation to the right was so marked that even after the application of cocaine no chink could be made out. The patient was operated on (septum resection), and the postoperative readings are given for comparison.

CASE 5 (Tully).—Marked right septal deviation in an emphysematous patient (fig. 5). The patient was hit by an automobile eight months previously, since

when he had found it hard to breathe through the right nostril, and for the past three months he had also complained of "stuffiness" and impaired hearing on the right side.

CASE 6 (Settleman).—Alternating nasal obstruction with pressure changes (fig. 6). The patient had had difficult nasal breathing which alternated from one side to the other. At the time of examination the right side was blocked.

The right nasal fossa was closed due to a turgescent inferior turbinate, and there was edema over the lower septum with some deviation posteriorly to the right. The left side was more patent and presented a large septal spur inferiorly.

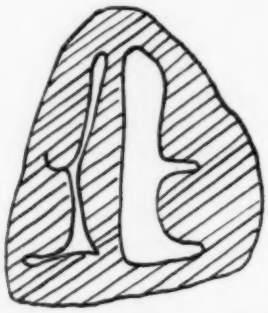


Fig. 4 (Harrison).—Chronic right nasal obstruction, structural. Objective obstruction and subjective obstruction; pressure tests confirmatory.

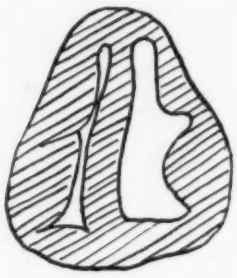


Figure 5

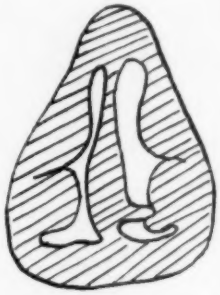


Figure 6

Fig. 5 (Tully).—Right complete nasal obstruction, structural. Objective obstruction and subjective obstruction; pressure tests confirmatory.

Fig. 6 (Settleman).—Slight nasal obstruction on right, alternating. Objective obstruction and subjective obstruction; pressure tests showed slight changes.

CASE 7 (Aronowitz).—Alternating nasal "obstruction" without pressure changes (fig. 7). For a number of years the patient had suffered from frequent "colds" in the nose, mainly during the winter, characterized by a watery nasal discharge. He also had alternating nasal obstruction. When lying in bed, he found breathing through the nose almost impossible and was therefore forced to keep his mouth open. He had had his tonsils removed eight years previously because of the nasal trouble and had recently been advised to undergo an operation on the nose.

The right nasal cavity was almost completely occluded by soft congestion and edema involving the mucous membrane over the turbinates, mainly the lower one, and the septum. The left fossa was also edematous, especially over the inferior turbinate, but not sufficiently to affect the patency of that side to any marked degree.

CASE 8 (Arick).—Acute traumatic right nasal obstruction (fig. 8). The patient had been punched in the nose two days previously, and the nose now felt "like it was paralyzed," and he could not breathe through the right side.

There was almost complete deviation of the septum to the right. The applicator could not be inserted beyond the deviation. After the application of cocaine to the right side, a small chink could be made out, but breathing was still difficult.

INTERPRETATION OF PRESSURE READINGS

CASE 1 (Myself).—While the forced pressure reading on myself registered higher values in expiration than in inspiration, it is significant that the right and the left sides were equal in either instance. As to

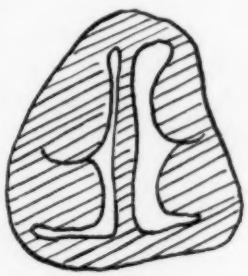


Figure 7

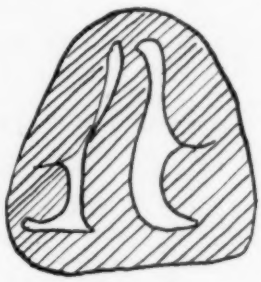


Figure 8

Fig. 7 (Aronowitz).—Alternating pseudo-obstruction on right. Objective obstruction and subjective obstruction; pressure tests normal.

Fig. 8 (Arick).—Acute right nasal obstruction, structural. Objective obstruction and subjective obstruction; pressure tests confirmatory.

the suction and buccal pressures, I am not as yet satisfied that they have any place in these tests, but merely include them for purposes of completeness.

The natural inspiratory and expiratory nasal records were also equal on the two sides.

It is interesting to observe the sharp difference between the natural and forced pressures, respectively. If one, for example, found the natural expiration to read 10 mm. of water and the forced nasal expiration 90 mm. of mercury, or 12,350 mm. of water pressure, one could express it in another way by saying that there is a latent reserve pressure of 12,340 mm. of water which the nose can bring into play if necessary. As to the significance of this differential force, I am not yet prepared to explain.

Pressure Readings in Eight Cases *

	Myself	Myself, Right Nostril Plugged with Cotton	Myself, Right Nostril Tightly Plugged	Prinz	Shaughnessy	Shaughnessy after Cocaine	Harrison	Harrison after Operation	Tully	Settleman	Settleman after Cocaine	Aronowitz	Arick	Arick after Cocaine
1. Complaint: side obstructed	Nil	Right	Right	Nil	Left	Nil	A.O.	Nil	Right	A.O.	Nil	A.O.	Right	Right
2. Objective: obstruction	Nil	Right	Right	Right	Left	Nil	Right	Nil	Right	Right	Nil	Right	Right	Right
3. Forced inspiration (in mm. Hg)														
A. Right nasal	70	70	**10**	50	30	25	**10**	25	**0**	50	50	60	**0**	**10**
B. Left nasal	70	70	70	50	**5**	**20**	50	30	15	50	50	60	30	40
C. Binasal	75	..	..	60	20	25	50	30	15	50	50	60	30	30
D. Postoral	75	..	..	40	..	..	..	..	20	45	45	60	35	35
E. Suction	130	..	..	..	..	..	70	..	70	120	120	..	..	..
F. Difference right and left nasal	0	0	**60**	0	**25**	5	**40**	5	**15**	0	0	0	**30**	**30**
4. Forced expiration (in mm. Hg)														
A. Right nasal	90	90	60	60	45	45	50	25	**0**	45	45	70	30	35
B. Left nasal	90	90	90	60	45	40	60	20	20	45	45	70	50	45
C. Binasal	90	..	..	70	45	45	50	25	20	45	45	75	45	45
D. Oral	90	..	..	50	40	40	..	..	30	50	50	70	45	45
E. Buccal	120	..	..	80	..	..	70	..	..	70	70	..	..	..
F. Difference right and left nasal	0	0	**30**	0	0	5	10	5	**20**	0	0	0	**20**	**10**
5. Natural inspiration (in mm. H_2O)														
A. Right tubal	10-12	5	**30**	12	**30**	30	15	20	8	15	15	8	25	25
B. Left tubal	10-12	**60**	**>150**	12	**0**	20	**50**	20	**0**	**4**	15	10	**125**	**120**
C. Postoral	3-5	3-5	3-5	4	..	..	..	..	..	3-6	4-6	5-7	..	..
D. Difference right and left nasal	0	**55**	**>120**	0	**30**	10	**35**	0	**8**	**11**	0	2	**100**	**95**
6. Natural expiration (in mm. H_2O)														
A. Right tubal	10-12	5	25	10	**40**	20	**40**	10	8	**30**	20	10	20	20
B. Left tubal	10-12	**60**	**>150**	8	10	15	20	12	0	5	20	12	**60**	**50**
C. Difference right and left nasal	0	**55**	**>125**	2	**30**	5	**20**	2	**8**	**25**	0	2	**40**	**30**
7. Breaths/minute (natural method)	18-20	20	..	20	20	18	..	18-20	15-20	15-18	18-20	19	..	..
8. Pressure differences														
A. Forced pressures (in mm. Hg)														
1. Right inspiration and expiration	20	20	**50**	10	**15**	20	**40**	0	0	5	5	10	**30**	**25**
2. Left inspiration and expiration	20	20	20	10	**40**	20	10	10	5	5	5	10	**20**	5
B. Natural pressures (in mm. H_2O)														
1. Right inspiration and expiration	0	0	5	21	10	10	**25**	10	0	**15**	5	2	5	5
2. Left inspiration and expiration	0	0	..	4	10	5	**30**	8	0	1	5	2	**65**	**70**

* In the table the numbers in bold face type indicate significant variations; > indicates greater than, and A.O. indicates an alternating obstruction.

On plugging my right nostril with cotton, I found that the forced readings were not altered, except if the plugging was very tight, and then the effect was surprisingly little. There were, however, marked changes in the natural pressures, the left tubal being twelve times as much as the right tubal pressure. This is sufficient to point to differences in both sides of the nose.

By the "tight" method, whereby the nasal tip (cannula) is covered with a piece of adhesive plaster, I reproduced in myself changes that I found in marked nasal obstruction (as in the case of Arick), and as in the cases examined my inspiratory pressure was affected out of proportion to the expiratory pressure, indicating, as I have repeatedly observed, that with progressive nasal obstruction the forced inspiratory pressure is lowered, while the expiratory pressure may maintain normal proportions until the obstruction is practically complete.

The natural postoral pressures were quite low and did not vary much whether the nose was patent or completely obstructed on one side. From this observation and from studies on patients I am not yet convinced that this method, termed the posterior rhinomanometry of Beyne by Worms and Bolotte, is a dependable procedure as they confidently led me to believe at first.

It must again be emphasized that this is essentially a differential test, and that a forced reading of 70 or of 90 mm. of mercury is meaningless unless one side of the nose is compared with the other. Another subject with equal patency may register higher or lower figures, and here too there will be no differences in pressures on the two sides.

Case 2 (Prinz).—The readings in this case were essentially normal. While the forced nasal pressures registered lower values than mine, it was found that both sides were also equal and that the forced nasal expiratory force was somewhat higher than the inspiratory force, as in my case.

The small differences in the right and left tubal pressures, as well as the inspiratory and expiratory tubal pressures, appear without significance.

Here too the natural postoral pressures are quite low.

Case 3 (Shaughnessy).—In this case there were a few points of interest. While an actual nasal obstruction was found to exist, its manometric response as regards natural pressures was by no means uniform. For example, compare this case with that of Arick, also one of nasal obstruction. Whereas in the case of Arick the natural inspiratory pressure was much higher than the expiratory pressure, the exact reverse happened in this patient. The significant feature, however, was that there was a loss of equilibrium.

A disturbance in the forced pressures, when this exists, on the other hand, makes simple the localization of the side of obstruction. The forced inspiratory reading on the left side was disproportionately low and pointed to obstruction on that side.

After applying a cocaine-epinephrine solution to the nose, there was a notable tendency toward equalization of pressures on both sides, both in the forced and in the natural methods. In cases of bony obstruction, as in marked septal deviation, such changes would not be expected. It is interesting to observe in this and in other cases of almost complete obstruction that while the forced inspiratory pressure is sharply lessened, the forced expiration is not disturbed.

Case 4 (Harrison).—In this case, too, there was sufficient manometric confirmation of the rhinoscopic findings. The forced nasal inspiratory pressure was low on the obstructed side. This was not so with the forced expiration, which here too yielded a fairly good reading.

It should be observed that the natural nasal pressures were much higher than one would expect. They were much like the readings obtained in myself with one side of the nose tightly occluded.

About two weeks after resection of the septum, there was a marked tendency to equalization of the pressures. One should observe also that the highest levels obtained at this time were lower than before the operation. I cannot account for this, except possibly to attribute it to a postoperative asthenia (the patient was 58 years old) and to some residual edema.

Case 5 (Tully).—That the right-sided obstruction was absolute can readily be found in the zero forced expiratory pressure, which represents the terminal stage in the progression of a nasal obstruction. The left natural tubal pressure was zero on the side opposite to that obstructed, a not uncommon phenomenon. It is likely that another person with greater vital capacity than this patient would have been able to generate sufficient pressure to at least produce a forced expiratory reading. This subject was of a typical emphysematous type, with a ruddy alcoholic complexion, and besides had weathered an attack of pneumonia and complicating pleurisy one year previously. These facts would probably account for the low forced pressure reading on the normal left side, and in turn would explain the other changes.

Case 6 (Settleman).—In spite of the fact that there was evident nasal occlusion, more marked on the right side, the forced pressures were found to be equal on both sides, indicating a milder form of obstruction, if any. The natural pressures as shown by the water manometer, however, revealed distinct and appreciable changes in the inspiratory and expiratory phases on both sides. Following the application of a cocaine-

epinephrine solution, the pressures were equalized. Such a resumption of equilibrium following the application of an astringent points against bony hypertrophy or displacement.

CASE 7 (Aronowitz).—It is obvious from this patient's pressure tests that the obstruction about which he complained was apparent rather than real; otherwise, the natural pressures should at least have been affected. If nasal obstruction means pressure variations as shown in such instances as septum deviation or acute influenzal rhinitis, then the postural edemas of the nose should be classified under a different category, for example, pseudo-obstruction.

CASE 8 (Arick).—In this case, as in that of Harrison, the natural pressure records were quite high. One should observe the marked differences between the inspiratory and expiratory pressures on both sides, which are significant.

By the forced method the right nasal inspiration (obstructed side) was zero. That this obstruction was not absolute may be found in the forced expiratory pressure, which registered 30 mm. of mercury.

CONCLUSIONS

1. The dynamics of nasal function offers a vast field for clinico-physiologic research.

2. The rhinometric test is essentially differential. Figures are meaningless unless one can find appreciable or marked differences in both nasal passages.

3. A disturbance in the equilibrium of nasal pressures points to a mild or severe degree of nasal obstruction, depending on the extent of the changes in the natural and forced pressures, respectively. In cases of slight nasal obstruction, the forced nasal readings are usually normal, while the natural pressures show appreciable changes. Even moderate cases of nasal obstruction will not affect the forced readings. When the forced pressures are affected in the more severe types, the inspiratory pressure is the first to be diminished, and the expiratory pressure will continue to register normal or subnormal readings until the obstruction is almost complete. A progressive nasal obstruction, therefore, first affects the natural pressures, next, the forced inspiratory pressure and, finally, the forced expiratory pressure.

4. The forced readings are more conclusive in localizing the side of the obstruction and will be found to be less on the obstructed side, and one may reason inversely by stating that when the forced nasal pressures are disproportionately diminished on one side (as compared with the other) one is dealing with a case of homolateral nasal obstruction. With the natural breathing tests, on the other hand, the results

may be at variance with what one would expect, but may be sufficiently unequal to point to trouble on one or the other side.

5. Nasal obstruction, though not uncommon, is far less frequent than one would expect. This I have learned from an investigation in a busy nose clinic over an extended period. One too often records a symptom as nasal obstruction without going into further details.

6. Pressure tests can hardly find their place as a routine clinical procedure, mainly because they are too time-consuming. They should be of value:

(*a*) In doubtful or borderline cases.

(*b*) In convincing the patient that his nose is functioning properly from the dynamic standpoint by having him see for himself the equality of pressure on both sides.

(*c*) In recording the benefits that may accrue from an operation by postoperative records.

(*d*) In demonstrating to the student the changes that may occur with nasal obstruction.

(*e*) In furthering the cause of conservative surgical treatment.

7. Artificial nasal obstruction of varying degrees with corresponding pressure changes which compare favorably with what one would expect with clinical nasal obstructions may be successfully reproduced by simple artificial methods.

8. While a marked drop in the forced pressure points to nasal obstruction, one should bear in mind that asthenic states and pulmonary disease, such as emphysema, may render it impossible for the patient to generate a greater force. In these cases, too, the differential changes should guide one in determining the condition of the nose.

9. Oral records, by both the forced and the natural method, were not helpful in arriving at these conclusions.

Clinical Notes

ACUTE MASTOIDITIS WITHOUT INVOLVEMENT OF THE MIDDLE EAR OR THE ANTRUM

GILBERT J. ROBERTS, M.D., POMONA, CALIF.

Acute mastoiditis without an apparent otitis media has frequently been reported. The conclusion has been that most of these cases of acute mastoiditis arose from transient otitis media evidences of which had disappeared when the mastoiditis was recognized, the infection having passed via the tube, the middle ear and the aditus to the antrum. Then the aditus became choked off with thickened mucosa, allowing the infection of the middle ear to subside, while the infection persisted in the mastoid. If at operation no evidence of infection is found in the antrum, then one may question the possibility of the mastoid infection having arisen from the middle ear.

I have been able to find but two cases of this sort reported in the English and American literature. In 1911, Crockett [1] reported a case of acute mastoiditis with an extradural abscess over the cerebellum. There was no history of otitis media or evidence of infection in the middle ear or antrum. The same year Fleming [2] reported a case of otitis media without involvement of the antrum or the middle ear. This patient was under treatment for tuberculosis at the time and subsequently was found to have a positive Wassermann reaction.

The following case was observed in the Ear, Nose and Throat Department of the Stanford University School of Medicine.

REPORT OF CASE

M. L., a white girl, aged 6 years, was seen by me on Dec. 8, 1931, complaining of a swelling behind the right ear of three days' duration. There had been an attack of otitis media four years previously, with spontaneous rupture of the drum membrane, which subsided in five days. The present trouble began four days previous to entry with pain behind the right ear, and on the second day a swelling appeared over this region. There were no history of trauma and no symptoms referable to the middle ear, and there had been no aural discharge. The child did not appear ill, but she was pale and undernourished and had a temperature of 100 F. Otologic examination gave negative results except for a small swelling over the right mastoid which was tender. Both ears were dry, and the drum membranes appeared normal. There was no periostitis of the canal. The hearing was normal for both ears. Roentgenograms showed a diffuse cloudiness over the right mastoid. The following day the leukocyte count was 11,950, with 55 per cent polymorphonuclears, and the temperature was 99 F.

At operation a large defect was found in the cortex of the mastoid from which pus welled up, and a large cavity with pus and necrotic tissue was found occupying the mastoid process. The sigmoid sinus and a large area of the cerebellar dura were found exposed. No communication was found between this cavity and the

1. Crockett, E. A.: Atypical Mastoiditis, Laryngoscope **21**:763, 1911; Tr. Am. Laryng., Rhin. & Otol. Soc., 1911, p. 134.

2. Fleming, E. W.: Necrosing Mastoid, Without Involvement of Antrum, Middle of Internal Ear, South. California Practitioner **26**:186, 1911; Tr. Am. Laryng., Rhin. & Otol. Soc., 1911, p. 511.

antrum. The upper part of the mastoid was occupied by sclerotic bone. By working down from the zygoma along the tegmen, the antrum was entered without exposing the dura, and a large, dry cavity was uncovered which proved to be the antrum. A probe could be inserted into a large, patent aditus. The membrane lining the antrum and aditus was thin and pale and showed no evidence of abscess. The antrum cavity appeared to be walled off from the remainder of the mastoid by sclerotic bone. Cultures from the mastoid showed Staphylococcus aureus.

The wound healed promptly, and the child made a complete recovery.

COMMENT

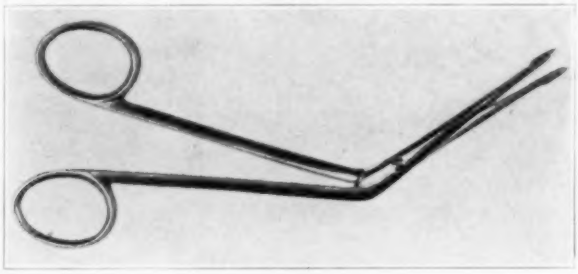
With a normal middle ear and antrum it seems unlikely that this extensive mastoiditis arose from a recent otitis media. Two possible explanations occurred to me: The first is that the mastoiditis was the result of a hematogenous infection, although at no time did the symptoms suggest an infection of the blood stream. The second possibility is that of a latent infection following the otitis media four years previously. The child's general condition before the present illness and the subsequent improvement might be considered in favor of this hypothesis.

In conclusion, I would point out that acute mastoiditis without recent disease of the middle ear is rare. Acute mastoiditis without involvement of the middle ear or the antrum is extremely rare.

MYRINGOTOMY SCISSORS

B. M. Becker, M.D., Brooklyn

The instrument is not unlike a fine aural forceps, with the essential difference that the cutting blades are confined to the ends of the instrument and are directed outward facing away from each other. The instrument is inserted through the most conspicuous bulging of the drum membrane, and then the blades are opened.



Myringotomy scissors.

The advantage over the use of the knife is that one is always assured of adequacy of the incision no matter at what point the instrument enters the drum membrane. This instrument provides greater safety and efficiency than the knife in paracentesis because of better control in its manipulation. The instrument is manufactured by Pfau's American Instrument Company, 2 East Twenty-Third Street, New York.

4802 Eighteenth Avenue.

From the Departments of Otolaryngology, the Long Island College and the United Israel-Zion Hospitals.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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ADVANCES DURING THE YEARS 1931 AND 1932 IN THE FIELD OF ALLERGY AS RELATED TO OTOLARYNGOLOGY

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The literature on the subject of allergy has become so voluminous that a review which approaches completeness far exceeds the scope of this article. The limited number of references here presented is intended to illustrate the drift of the newer work along this line.

The chief developments presented during the past year lie in the discovery of new agents which cause allergy, more complete studies of molds and fungi in relationship to allergy, more complete study of oily substances of plant origin in relationship to dermatitis and more numerous reports of the conditions which have been grouped under the term physical allergy.

MECHANISM OF REACTION

Cohen, Ecker and Rudolph [1] reported further results on a method of study which they introduced with Breitbart [2] in 1930. Their observations present a new idea concerning the fundamental cause of the hay fever and asthma reaction. The authors expressed the belief that the hay fever and asthma reaction, when caused by sensitiveness to pollen, dander or other inhalant substances, is analogous to the Arthus phenomenon and due directly to the fact that nature holds the offending substance locally on the membranes where it lights instead of allowing it to be absorbed and carried away by the blood. When an allergen is held locally it does more harm at that site than if it were absorbed and carried away, but, at the same time, it is less likely to cause a general reaction. In other words, the offending allergens are excluded from the blood stream and are not allowed to reach the tissues generally.

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1. Cohen, M. B.; Ecker, E. E., and Rudolph, J. A.: The Effect of Allergy on Allergen Absorption Following Parenteral Injections, J. Allergy **2**:453, 1931.

2. Cohen, M. B.; Ecker, E. E.; Breitbart, J. R., and Rudolph, J. A.: The Rate of Absorption of Ragweed Pollen Material from the Nose, J. Immunol. **18**: 419, 1930.

Cohen and his associates were able to demonstrate ragweed pollen products in the blood of normal persons during the ragweed season, but they were unable to demonstrate even a trace in pollen-sensitive persons. Furthermore, even in timothy-sensitive persons who were not sensitive to ragweed pollen, they were unable to demonstrate ragweed products in the blood at the height of the ragweed season, nor even when ragweed products were sprayed directly into the nose, in spite of the fact that ragweed pollen, when sprayed into the nose, would not cause a reaction in timothy-sensitive persons. Further results showed that in dust-sensitive persons who did not react to ragweed, the subcutaneous injection of pollen in large quantities is not followed by the appearance of ragweed products in the blood stream. The pollen is apparently held locally by the tissues. Pollen can be found with ease in the blood of normal persons after similar hypodermic injections.

Cohen and his co-workers reported that their observations harmonize with those of Opie [3] obtained in the study of the Arthus phenomenon caused by serum. Opie found that in animals sensitized to foreign serum, the tissues were inclined to hold the serum locally on subsequent inoculations; in fact, when subsequent inoculations of serum caused necrosis and gangrene, Opie could find the injected material in the gangrenous areas but not in the animals' blood.

If the conclusions of these observers are correct, the hay fever and asthma reaction in the case of sensitiveness to inhalant substances is not altogether a constructive phenomenon of nature, because it does not confer general immunity as it might if it were absorbed. The failure of the tissues to absorb offending allergens protects the patient in a way from general reaction but does so at the expense of a damaging reaction in the tissues locally in the nose and bronchial tubes.

Smyth and Stallings [4] added an interesting elaboration to the earlier work of Walzer and Walzer [5] in showing that undigested egg can be absorbed from the gastro-intestinal tract in normal persons and cause typical hives in passively sensitized areas of the skin. This work is directly contrary to the older views of physiologists who believed that the gastro-intestinal membranes excluded undigested proteins from absorption. The older views of the physiologists are only grossly correct. The work of Walzer and Walzer and that of Smyth and Stallings

3. Opie, E. L.: Relation of Antigen to Antibody (Precipitin) in Circulating Blood, J. Immunol. **8**:55, 1923; Fate of Antigen (Protein) in Animal Immunized Against It, J. Exper. Med. **39**:659, 1924.

4. Smyth, F. S., and Stallings, Minnola: Studies in Passive Transfer: 4. Rectal Absorption of the Antigen (Egg White), J. Allergy **3**:16 (Nov.) 1931.

5. Walzer, A., and Walzer, M.: Urticaria: II. The Experimental Wheal Produced on Normal Skin Through Internal Changes, Arch. Dermat. & Syph. **17**:659 (May) 1928.

prove that undigested albumins can be absorbed in traces sufficient to cause allergy in sensitive persons. The work of the latter authors was proved through the taking of blood from an egg-sensitive person and its injection intracutaneously into normal persons and, at a later time, the injection of egg solution into the rectum. In their experiments, hives were found to appear within from twenty to sixty minutes in the skin sites locally sensitized to egg.

Ratner,[6] in reporting further studies on the transmission of hypersensitiveness to certain substances from mother to offspring, found evidence to prove that the antibodies of the mother are often transmitted to the offspring in such quantity that the infant's serum, when injected into guinea-pigs, sensitizes the animal to the agent to which the infant reacts.

This type of sensitiveness, which is passive, is quite different from the active type, which the same infant may acquire by true mendelian heredity. An infant's reaction to certain substances acquired passively in this way is identical with that of the mother. However, the infant who inherits passive sensitiveness does not react to all the substances to which the mother is sensitive. On the other hand, the infant, at the time of testing, may react to antibodies which do not afflict the mother.

Sherrer,[7] in an endeavor to discover the whereabouts of the specific reagins in cases of hay fever and asthma, separated a number of bodies out of the plasma and found that the reagins follow the globulin fraction of the blood proteins. The reagins of allergy, therefore, are similar in this respect to other immune bodies, including diphtheria antitoxin.

Bowman and Walzer[8] studied the effect of local allergy reactions on subsequent reactions in the same site caused by the same and different substances. When a solution of codeine is injected intracutaneously into the skin of a normal person, it reacts locally with the formation of a large wheal. If the same material is injected into the same skin site at a later period, the wheal will reform, but, according to Bowman and Walzer, to a much less marked degree during a period that may last from several days to several weeks. In other words, the authors found that the skin loses its capacity for reaction by reacting.

Vallery-Radot, Mauric, Hugo and Giroud[9] attempted passively to transfer the sensitiveness of human allergy into animals by choosing

6. Ratner, Bret, and Greenburgh, J. E.: Congenital Protein Hypersensitiveness: Protein Hypersensitiveness Transmitted from Allergic Mother to Child, J. Allergy **3**:149 (Jan.) 1932.

7. Sherrer, E. L.: The Distribution of Reagins in the Blood Plasma, J. Allergy **2**:467, 1931.

8. Bowman, Katherine, and Walzer, Matthew: A Study of the Refractoriness to Whealing in Normal (Nonatopic) Skin, J. Allergy **3**:503, 1932.

9. Vallery-Radot, P.; Mauric, G.; Hugo, A., and Giroud, P.: Passive Anaphylaxis: Injections into Rabbits of Human Serum from Hypersensitive Individuals, Mixed with the Specific Allergen, Compt. rend. Soc. de biol. **107**:797, 1931.

patients who had demonstrable reagins in their blood, mixing their serum with the offending allergens and injecting this into animals. Fifteen experiments were tried in this way and gave negative results in each instance. They assume that passive transfer of allergy to animals cannot be accomplished through the use of mixtures of reagins and allergens.

Walzer,[10] in studying passive transfer from more than 200 cases of atopic hypersensitiveness, found 3 patients whose skin reacted slightly or markedly positively to practically every substance used for test material.

Strange to relate, the serums of these three persons differed from all the others in that when injected into the skins of normal persons they passively sensitized the skin to practically every material which was injected, even substances such as physiologic solution of sodium chloride, distilled water and normal human serum. Walzer thought that this type of serum carried with it a capacity for nonspecific sensitization.

Sulzberger,[11] in studying the transferability of sensitiveness to Trichophyton, found that some persons will and some will not accept passive transfer. He found, also, in testing patients with injections of Trichophyton, that they reacted alike to extracts prepared from several varieties of Trichophyton, Achorion, Epidermophyton and Microsporon.

STUDIES OF THE CAUSES OF ALLERGY

Dander and Pollen Atopens.—Stull, Cooke and Chobot[12] isolated water-soluble, albuminous proteins from long and short ragweed extracts which were found to be chemically similar in nature and biologically identical. The purified protein from the short ragweed was as effective in the treatment of hay fever as the extract from giant ragweed.

These results are in harmony with the view expressed previously by Cooke and Vander Veer,[13] Coca and Grove[14] and Prausnitz and

10. Walzer, Matthew: A Non-Specific Cutaneous Sensitivity which is Passively Transferable with the Serum to the Normal Human Skin: Preliminary Report, J. Immunol. **23**:99 (July) 1932.

11. Sulzberger, M. B.: Experiments in Passive Transference of Urticarial Hypersensitiveness to Fungous Extracts: (*A*) Immunological Grouping of Trichophytons and Monilia; (*B*) Refractoriness of Certain Individuals to Sensitization by Reagins, J. Immunol. **23**:73 (July) 1932.

12. Stull, Arthur; Cooke, R. A., and Chobot, Robert: The Identity of the Allergically Active Substance in the Giant and Low Ragweed Pollen, J. Allergy **3**:120 (Jan.) 1932.

13. Cooke, R. A., and Vander Veer, A.: Human Sensitization, J. Immunol. **1**:201, 1916.

14. Coca, A. F., and Grove, E. F.: Studies in Hypersensitiveness: Study of Atopic Reagins, J. Immunol. **10**:445, 1925.

Küstner,[15] that the active exciting substance in the two weeds is the same.

Stull, Cooke and Chobot,[16] in a later communication, stated that the active substance of timothy protein is an albuminous protein similar to the active material extracted from ragweed pollen. They stated that it is shown to be the only active substance.

Alcoholic treatment of timothy pollen lessened the amount of extractable nitrogen and lessened its activity. Heating also lessened the activity of the extract.

The globulin content of the pollen and the reducing substance obtained from pollen showed no specific activity. Precipitin tests made with purified albumin and specifically sensitive serums were negative.

Stull and his co-workers found no albumin in cattail pollen, which they believe explains the absence of a clinical and a cutaneous reactivity to this pollen.

Stull, Cooke and Barnard [17] stated that after a clinical experience of four years with different varieties of grass pollen, they believe that the active substance in grass pollen is a single substance and is common to all extracts.

Kahn and Grothaus [18] reported that red cedar and mountain cedar have a cross-specificity; that is, patients in the environment of mountain cedar who have symptoms during December and January give positive skin reactions also to red cedar to which they are not exposed and can be relieved clinically by subcutaneous treatment with red cedar pollen.

Murphy and Cobe [19] found that through the hydrolysis of human dander a large quantity of histamine and histidine could be obtained. They compared the reactions obtained in a group of allergic and in a group of nonallergic patients tested intracutaneously with solutions of histamine of known origin and the histamine obtained through the treatment of human dander.

The percentage of positive reactions obtained by the histamine solution and the dander extract was very much alike in both groups

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15. Prausnitz, C., and Küstner, H.: Studien über die Ueberempfindlichkeit, Centralbl. f. Bakt. (Abt. 1) **86**:160, 1921.

16. Stull, Arthur; Cooke, R. A., and Chobot, Robert: The Allergically Active Substance in Pollen: A Chemical and Biologic Study of Phleum Pratense (Timothy) Pollen, J. Allergy **3**:341 (May) 1932.

17. Stull, Arthur; Cooke, R. A., and Barnard, J. H.: The Biologic Identity of Certain Grass Pollens Causing Hay Fever, J. Allergy **3**:352 (May) 1932.

18. Kahn, I. S., and Grothaus, E. M.: Hay-Fever and Asthma Due to Red Cedar (Juniperus Virginiana) and to Mountain Cedar (Juniperus Sabinoides), South. M. J. **24**:729, 1931.

19. Murphy, J. A., and Cobe, H. M.: Histamine: The Reactive Substance in Human Dander, J. Allergy **3**:161, 1932.

of patients, but the reactions were more strongly positive and persisted for a longer period in the allergic group.

Parlato [20] reported a third study of emanations from flies as an exciting cause of allergic coryza and asthma. Parlato's original paper, published in 1929, was directed toward a study of the sand flies which occur in swarms during the summer months in the Great Lakes' region. The insect wings are covered with tiny scales, which he found were liberated in the air and occurred in about the same concentration on pollen plates as the pollen granules themselves. He found patients who had coryza and asthma on exposure to the scales of these insects, who gave positive skin reactions to the extracts of these insects and who were therapeutically relieved by treatment with such extracts. Parlato broadened his study to include insects of the moth and butterfly group, and he found that they have an identical effect on sensitive patients from the standpoint of exposure to the scales, skin tests and results of treatment. He believes that the exciting agent in the scales of sandflies is identical, or, at least, closely related to that of moths and butterflies.

Sellers and Adamson [21] tested a group of 36 patients with the pollen of 6 different species of the chenopod group of weeds. A patient who reacted positively to one member of this group reacted positively to the other members with few exceptions. The authors believe, therefore, that they have established a cross relationship between the atopens of the different members of the group of chenopods.

They have substantiated the foregoing idea through experiments on passive transfer and desensitization and have found that the atopens of all 6 species are contained in the pollens of Amaranthus spinosus, Amaranthus palmeri, Amaranthus retroflexus and Acnida tamariscina, but that the atopens of the entire group are not contained in the pollen of Salsola pestifer or of Chenopodium album.

The foregoing study suggested that one of the members of the family of Amaranthus can be used for the testing of patients sensitive to 1 or all of the other 6 species.

Molds and Fungi.—Towey, Sweany and Huron [22] presented a new factor in the etiology of asthma through the discovery of fungus spores

20. Parlato, S. J.: The Sand Fly (Caddis Fly) as an Exciting Cause of Allergic Coryza and Asthma: II. Its Relative Frequency, J. Allergy **1**:307, 1930; A Case of Coryza and Asthma Due to Sand Flies (Caddis Flies), ibid. **1**:35, 1929; Emanations of Flies as Exciting Causes of Allergic Coryza and Asthma: III. Hypersensitiveness to Moths and Butterflies (Lepidoptera), ibid. **3**:125 (Jan.) 1932.

21. Sellers, E. D., and Adamson, W. B.: A Study of the Apparent Atopic Similarity of Certain Chenopodiales Pollens, J. Allergy **3**:166, 1932.

22. Towey, J. W.; Sweany, H. C., and Huron, W. H.: Severe Bronchial Asthma Apparently Due to Fungus Spores Found in Maple Bark, J. A. M. A. **99**:453 (Aug. 6) 1932.

in maple bark which affect a surprisingly large percentage of people who are exposed to it.

The patients studied, numbering 35 among workers in a saw mill and 10 among workers in a railroad tie factory, were found to have asthma when exposed to the dust from the dry bark of maple logs. They became free from asthma on avoidance of this exposure.

The offensive agent appeared as a sootlike material under the bark, which was identified by Dr. Thom, principal mycologist of the Bureau of Chemistry and Soils at Washington, as a fungus called Coniosporium corticola.

The history of the cases is interesting. Invariably symptoms appeared during the summer months in workmen who were exposed to the dust from maple logs. The illness started as a cough and shortness of breath of rather acute onset. The cough was associated with sputum, followed by fever, night sweats, loss of weight and substernal pain. The condition continued and got worse so long as the men were exposed to dust from the logs, but began to improve promptly if they were removed from contact with the logs.

Roentgenograms of the lungs showed definite organic lesions, consisting of thickening of the bronchi, especially in the lower lobes, associated with small flecking in the parenchyma of the lungs which resembled that of bronchopneumonia. The apexes of the lungs were invariably clear.

Skin tests with a suspension of the spores or an extract gave positive reactions not only in the afflicted persons, but also in those who were adequately exposed to the dust but who did not present asthmatic symptoms. Tests made with the same material on nonexposed persons gave negative reactions.

The results showed that a high ratio of the persons exposed to the dust acquired the spore infection. Towey and his associates believe that the pulmonary symptoms are due partly to a local toxic effect of the spore and partly to a foreign body reaction resembling protein sensitization in its clinical and immunologic aspects. They were able to prevent the condition by having the saws sprayed with water so as to prevent the scattering of the maple bark dust throughout the mill. They advise that workers in saw mills of this type be removed to another locality immediately if they show evidence of respiratory trouble.

The foregoing condition has to be separated in its etiology from the hereditary type of atopic sensitiveness, since the afflicted persons represent too large a percentage of those who are exposed. Furthermore, they did not give the positive family history or eosinophilic secretions typical of true atopic hypersensitiveness. The authors are inclined to

compare this illness with the illness discovered by Figley [23] among persons exposed to castor bean dust. This work ranks with Figley's as a banner contribution for the year in which it was published.

Jimenez-Diaz, Cuenca and Puig [24] discussed climatic asthma, and in trying to account for the change which occurs in apparently climatic cases, they emphasized the importance of the presence of pollen, fungi and other allergens which exist in certain localities and not in others. They tend to minimize the effect of temperature and air moisture, even in the condition they call coastal asthma, in which patients suffer attacks while at the seashore and improve on going inland. They believe that improvement is due to the escape from certain fungi and other allergens in household articles.

The authors studied the rôle of fungi extensively and expressed the belief that those principally responsible for the so-called house asthma are Aspergillus, Mucor, Pencillium and, to a lesser extent, Clamidoes. They studied these from the standpoint of the finding of fungi in the patient's environment and the finding of specific skin tests, by demonstration of passive transfer and by the provocation of attacks by the introduction of fungi into the patient's environment.

Cobe [25] reported a case of violent asthma caused by a mold (Cladosporium fulvum) which grows on tomato vines.

The patient gave a history of asthma which occurred coincident with the growth of this mold on tomatoes in greenhouses. His work brought him in constant contact with the mold.

The mold was isolated in culture and an extract made of it. The patient gave a positive skin reaction to a culture of the mold but negative reactions to an extract of the normal tomato leaf and negative reactions to other molds. Treatment with an extract of the mold resulted in such improvement in the patient that he could handle tomato vines from then on without ill effect.

Brown,[26] in discussing sensitization to fungi, stated that microorganisms may be divided into four groups: the molds, yeasts, protozoa and bacteria. The molds can be classified into thousands of different species. The fungi that are important from the standpoint of atopy, according to Brown, are yeasts, rusts, smuts and mushrooms. Different types of fungi can be cultured from the skin, sputum, nasal secretion, stools and dusts.

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23. Figley, K. D., and Elrod, R. H.: Endemic Asthma Due to Castor Bean Dust, J. A. M. A. **90**:79 (Jan. 14) 1928.

24. Jimenez-Diaz, C.; Cuenca, B., and Puig, J.: Climatic Asthmas, J. Allergy **3**:396, 1932.

25. Cobe, H. M.: Asthma Due to a Mold, J. Allergy **3**:389, 1932.

26. Brown, G. T.: Sensitization to Fungi, J. Allergy **3**:522, 1932.

Brown expressed the belief that hypersensitiveness to fungi may take its place with hypersensitiveness to pollens, epidermal materials, foods, etc., as a cause of asthma and eczema. Brown stated that fungi should be looked on as weeds of the home, and that specimens of dust taken from homes and mattresses are always found to contain fungi. These statements are in line with the idea of other workers in this field, who find that spores from molds are responsible for more cases of hay fever, asthma and dermatitis than was formerly believed. Brown used extracts from mold culture in therapy in much the same way that he did extracts from pollen in pollen therapy and obtained about the same result as is generally obtained in cases of sensitiveness to pollen.

Flood [27] reported a study of testing with 8 or 9 extracts of fungi on dust-sensitive patients. Whereas 8 patients gave positive skin reactions to the fungi, only 1 reacted to the fungus when it was sprayed into the throat.

White and Taub [28] reported 4 cases of fungus infection in which true allergic sensitiveness to other materials was found. When the patients avoided contact with these substances, which proved to be cotton seed oil, oatmeal, buckwheat and silk, respectively, the fungus infection could be controlled.

The conclusions were drawn that in some people with fungus infection the condition of the skin is so altered that it reacts to certain foods, and that improvement can be obtained by removal of contact with the offending agent.

Sensitiveness to Oily Substances.—Pilot [29] reported a case of chrysanthemum dermatitis in a florist, in which the leaves of the plant were the irritating factors. A patch test made with an ether-soluble fraction of the leaf gave positive reactions. Passive transfer tests were unsuccessful. He considered the condition similar to poison ivy dermatitis.

It may be mentioned in passing that sensitiveness to chrysanthemums is important not only in the case of florists, but also in the case of sensitive persons who are exposed to insect powders, such as pyrethrum.

Sulzberger and Morse [30] reported 2 cases of eczema caused by sensitiveness to wool fat. The use of ointments containing wool fats as a

27. Flood, C. A.: Observation on Sensitivity to Dust Fungi in Patients with Asthma, J. A. M. A. **96**:2094 (June 20) 1931.

28. White, C., and Taub, S. J.: Sensitization Dermatoses of Nonfungous Nature, J. A. M. A. **98**:524 (Feb. 13) 1932.

29. Pilot, Isadore: Dermatitis Venenata Due to Chrysanthemums, J. Allergy **3**:404, 1932.

30. Sulzberger, M. B., and Morse, J. L.: Hypersensitiveness to Wool Fat, J. A. M. A. **96**:2099 (June 20) 1931.

base made them distinctly worse. Strict avoidance of wool fat resulted in relief. Patch skin tests made with wool fat gave positive results.

Gay and Ketron,[31] in studying a case of ragweed dermatitis of the exposed parts, made the following observations:

Dry ragweed pollen, when applied to the skin, gave rise to an eczematous area at the site of application after a period of seventy-two hours.

Concentrated pollen extract, when applied to the skin, also gave rise to an eczematous area at the site of application.

Ragweed oil, similarly applied, gave positive patch tests.

Passive transfer tests with the serum of the patient gave positive reactions.

The authors were unsuccessful in producing dermatitis by repeated intracutaneous injections of the patient's serum into normal skin followed by the application of pollen oil to the treated areas. Pollen extracts likewise failed to produce dermatitis as a response.

When the serum of the patient was mixed with ragweed pollen oil and applied to the normal skin, it gave no reaction after a period of four days.

Flax.—The frequency with which intensely positive tests with cottonseed and flaxseed extracts are obtained in routine skin testing was overlooked to a large extent by the earlier workers on allergy as indicating a positive cause of clinical hay fever and asthma. Many of the more recent workers on allergy have pointed out the wide sphere of usefulness of cottonseed and flaxseed products, and through this they have been able to relieve a relatively large proportion of patients giving skin reactions to flax or cottonseed by removal of contact with one or the other of these two almost universally distributed substances. A smaller number of favorable results have been reported following specific treatment with one or the other of the two extracts. It may be said in passing that each of these two extracts has been avoided in therapy by a large number of specialists on allergy because of their high grade of toxicity.

Grant [32] stated that in routine tests on 111 patients, 6 gave marked positive reactions to flaxseed by the scratch method. Five of the patients in this group had asthma; 3 of them obtained complete relief from asthma by avoiding contact with flaxseed products, while a fourth obtained complete relief from desensitization therapy.

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31. Gay, L. N., and Ketron, L. W.: A Case of Ragweed Dermatitis Recurring Over a Period of Thirteen Years, J. Allergy **3**:478, 1932.

32. Grant, L. R.: A Report of Six Cases of Flaxseed Sensitization with Review of the Literature, J. Allergy **3**:469, 1932.

Grant mentioned the frequency with which flaxseed cases are found among people living in rural districts and who are in contact with animal feed and chicken feed.

Grant presented a most complete review of the literature on the subject of flaxseed as a cause of sensitiveness and clinical symptoms such as hay fever, asthma and dermatitis and discussed the extensive distribution of flaxseed meal as used for animal feed and even for human beings in the form of cereals. He also mentioned the widespread use of the oil and fiber of flax, and referred to its use in beauty parlors as a dressing for the hair.

Grant referred to the interesting contributions made by several observers concerning the commonness of dermatitis in workers in linseed oil factories. The workers are so prone to become eczematous after prolonged contact that the reaction cannot be considered specific but rather as due to a natural irritating effect of the material. Eczema of this type can be relieved by avoidance of contact for several days. It can also be relieved to a degree by having the workers wash their hands carefully with soap and water after prolonged contact.

Grant stated that the reactions in the gross majority of sensitized cases are caused by flaxseed itself rather than by the oil, linen or substances made from flax fiber. He stated that good results can be obtained in therapy by avoidance of flax if this is possible and by specific therapy when avoidance is not practical.

Bacteria.—Cooke [33] discussed infective asthma and found that in the truly infective cases, which he called nonsensitive, 45 per cent had an antecedent history of allergy. This he compared with the 52 per cent of heredity obtained in truly atopic cases. He found the eosinophilia characteristic of atopy just as typical of the infective cases as of the sensitive cases. He expressed the belief that asthma of infective origin should be considered an allergic reaction.

Drug Allergy.—The usefulness of ephedrine as an internal remedy is seriously impaired by the frequency with which certain pharmacologic effects of the drug produce disagreeable symptoms, such as nervousness, insomnia, weakness and palpitation. The ill effect can be reduced by giving the drug in the form of a solution instead of capsules and by reducing the dose to a point below that which produces disagreeable pharmacologic effects and yet serves to have a useful therapeutic effect. It is not always possible to arrive at such a happy balance between the good and the bad effects of ephedrine. In addition to the untoward pharmacologic effects of the drug, a number of cases have been reported in which persons responded to the drug with true symptoms of allergy.

33. Cooke, R. A.: Infective Asthma: Indication of Its Allergic Nature, Am. J. M. Sc. **183**:309, 1932.

This sphere of knowledge is useful to otolaryngologists because frequently an ill effect from this cause may result from the traces of the drug used in nasal sprays.

Scheer,[34] in 1929, reported a case of dermatitis of the face and the hands after the use of an oil spray containing ephedrine. Ramirez and Eller [35] reported 4 cases of contact dermatitis caused by sensitiveness to ephedrine.

Ayres and Anderson [36] reported 2 cases of dermatitis caused by the use of a nasal spray containing ephedrine. Their patients obtained relief on discontinuance of the spray. Positive skin reactions were obtained in 1 case by the application of ephedrine solution by scratch test, and in the other a markedly positive reaction was obtained by a patch test.

Bullen, Francis and Parker [37] reported 2 cases of sensitiveness to ephedrine with dermatitis which gave positive patch tests with a solution of ephedrine, characterized by redness, itching and vesiculation. Efforts at passive transfer were negative.

It is true, in general, that agents which relieve asthma symptomatically can sensitize the patient. It is strange, however, that direct antagonists of the allergy reaction, such as ephedrine and pituitary extract, should sensitize, and one cannot help questioning whether or not the result obtained is due to an impurity in the drug. This is as yet unproved pro or con. Several attacks of generalized allergy caused by pituitary extract are so well known that most physicians experienced in the treatment of allergy hesitate to use it without preliminary tests. Koessler thought that reactions of the sort were due to an admixture of histamine as an impurity in pituitary extracts.

I [38] reported 2 cases of drug allergy, 1 caused by sensitiveness to chlorine and 1 caused by sensitiveness to ether. The patients had coryza, cough and asthma on inhalation of the fumes referred to and gave positive immediate wheals without pseudopods after application of the offending substance to the skin. They apparently were not sensitive to other agents which were used in testing by the scratch and patch methods. The condition could not be transferred passively in either case.

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34. Scheer, M.: A Case of Dermatitis Venenata Due to Ephedrine, Arch. Dermat. & Syph. **20**:641 (Nov.) 1929.

35. Ramirez, M. A., and Eller, J. J.: Intradermal, Scratch, Indirect and Contact Tests in Dermatology, J. A. M. A. **95**:1080 (Oct. 11) 1930.

36. Ayres, S., Jr., and Anderson, N. P.: Dermatitis Medicamentosa Due to Ephedrine, J. A. M. A. **97**:437 (Aug. 15) 1931.

37. Bullen, S. S.; Francis, Nathan, and Parker, J. M.: Dermatitis Medicamentosa Due to Ephedrine, J. Allergy **3**:485, 1932.

38. Duke, W. W.: A Type of Drug Allergy Caused by Contact Sensitiveness to Ether and Chlorine, J. Allergy **3**:495, 1932.

Grishaw [39] found, in a case of severe urticaria and angioneurotic edema, that soon after injections of insulin the patient had positive skin reactions and severe clinical symptoms with two different preparations of insulin but not with a third preparation. The third preparation could be tolerated.

Efforts at passive transfer of this type of sensitization gave negative results. Grishaw assumed that the patient was not sensitive to insulin itself but to some undetermined product in particular preparations of insulin. This case seems analogous to the case of a physician of my acquaintance, who reacted with severe allergic manifestations to only one of five different preparations of acetylsalicylic acid.

Effect of Diet on Reaction.—In the study of a person sensitive to codfish Stoesser [40] found that when the patient was on a ketogenic diet with a normal intake of fluid, no protection against asthmatic attacks was noticeable. Furthermore, a high carbohydrate diet gave little protection.

When the intake of water was low, with the patient on a ketogenic diet, the reaction to codfish was worse. The same was true when the patient was on a high carbohydrate diet.

When the patient was taking large quantities of fluid with a low carbohydrate diet, full protection was obtained if the patient was quiet and remained indoors. This protection failed, however, when the patient was active.

The administration of dextrose intravenously would stop an attack. Stoesser draws attention to the fact that the much used epinephrine raises blood sugar, but he fails to draw conclusions concerning this relationship without further study.

DIAGNOSTIC METHODS

Peshkin [41] reported a study of an ophthalmic test made with dry pollen in cases of hay fever and asthma in which, although diagnosed definitely as pollen cases, the patients failed to give positive skin reactions and also failed to give positive reactions to ophthalmic tests made with solutions of the suspected pollens. A certain number of these patients who gave positive reactions with the dry pollen not only gave negative skin reactions but also negative reactions to ocular tests with pollen solutions. Peshkin offers this as an additional method of testing for the positive diagnosis of pollen disease.

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39. Grishaw, W. H.: Allergic Manifestation to Insulin, J. A. M. A. **97**:1885 (Dec. 19) 1931.

40. Stoesser, A. V.: Recent Observations in the Study of Asthma Due to Food Allergy, J. Allergy **3**:332, 1932.

41. Peshkin, M. M.: XI. A Dry Pollen Ophthalmic Test in Pollen Asthma and Hay Fever Patients Negative to Cutaneous Tests, J. Allergy **3**:20 (Nov.) 1931.

Peshkin's method of testing cannot be used as a routine measure, but should be extremely valuable in testing patients with pollen cases who give negative skin reactions and negative reactions to ophthalmic tests made with solutions.

Alexander [42] reviewed the results of skin tests in 11,443 cases of allergy reported by 44 different observers. Positive skin reactions to one substance or another were found in the following percentages of the following illnesses: hay fever, 93.2; vasomotor rhinitis, 55.7; eczema (infantile and adult), 52.7; bronchial asthma, 52.7, and gastro-intestinal allergy, 26.5.

Of reports on 32,182 tests, exclusive of pollens, the following results were obtained:

Of the inhalant group, feathers gave 20.3 per cent positive reactions, or about one positive for every five tests made with feathers. In the order named, the reactions were as follows: orris root, 16.9 per cent; horse dander, 16.6 per cent; wool, 11.3 per cent; pyrethrum, 9.3 per cent; cat dander, 9.2 per cent; cattle dander, 8.7 per cent, and dog dander, 8.3 per cent. All other inhalants gave less than 5 per cent. Of the foods in order, wheat gave 22.4 per cent; egg, 18.4 per cent; milk, 14.8 per cent; chocolate, 13.9 per cent; spinach, 13.3 per cent; beans, 12.9 per cent; potato, 12.1 per cent; tomato, 11.9 per cent; carrot, 11.8 per cent; pea, 11.5 per cent; barley, 10.7 per cent; rye, 8.9 per cent; pork, 8.3 per cent; beef, 7.7 per cent; oats, 6.7 per cent; corn, 5.7 per cent, and rice, 5.7 per cent; all others gave less than 5 per cent. It is noteworthy that the reactions to spinach, tomato, carrot, pea, barley, oat and rice appeared mostly in cases of infantile eczema. Pepper gave 17.5 per cent, but notations were made frequently that this may be a nonspecific reaction.

Cohen and Rudolph,[43] in a differential diagnosis between allergy and infections of the respiratory tract in children, stated that the allergic conditions are characterized by frequent recurrence, lack of evident contagion, a pale appearance of the membranes and the finding of an eosinophilia amounting to 10 per cent or more in the nasal secretions. The authors warn against conclusions being drawn from cloudiness of the sinuses in roentgenograms and dependence on skin tests.

Fineman [44] found that in the diagnosis of asthma in patients who failed to show expiratory râles at the time of the examination, râles could be elicited through the application of pressure to the chest followed

42. Alexander, H. L.: An Evaluation of the Skin Test in Allergy, Ann. Int Med. **5**:52, 1931.

43. Cohen, M. B., and Rudolph, J. A.: Allergic and Infectious Conditions of Upper Respiratory Tract in Children, J. A. M. A. **97**:980 (Oct. 3) 1931.

44. Fineman, A. H.: The Pressure Test as an Aid in the Clinical Diagnosis of Bronchial Asthma, M. J. & Rec. **135**:425 (May 4) 1932.

by deep inspiration. He expressed the belief that through the use of forced expiration applied in this way, an additional number of cases can be positively diagnosed by oscultation.

EFFECTS OF ALLERGY

Dermatitis.—Sulzberger, Spain, Sammis and Shahon,[45] after studying hypersensitiveness in certain types of dermatoses, made the following report. They stated, with reason, that the term eczema is a scrap basket term under which many different skin diseases have been grouped. They prefer, with the European workers in this field, to confine the term to the group of dermatoses caused by contact irritants. This type of dermatitis is usually confined to the exposed parts, consists of erythema, papules, scaling and vesiculation and is often caused by contact irritants of nonprotein nature. It is commonly caused by oily constituents of plant origin, such as poison ivy, primrose and ragweed oil and finally, by drugs and chemical bodies contacted vocationally. It is interesting to mention that the patients usually give a family and personal history which is negative for allergy and negative for infantile eczema. They usually give positive reactions to patch tests with offending excitants, and negative reactions to passive and intracutaneous tests.

For routine patch testing, Sulzberger and his co-workers used the following list of substances:

Contact Irritants

Poison ivy (1:5).	Potassium iodide ointment (25 per cent).
Procaine hydrochloride (1 per cent).	Iodoform (powder).
Metaphen (standard solution).	Corrosive mercuric chloride (1:1,000 solution).
Mercurochrome-220 soluble.	Naphthalene (1 per cent solution in alcohol).
Nicotine salicylate (10 per cent).	Formaldehyde (5 per cent).
Tincture of pyrethrum (standard solution).	Nickel sulphate (5 per cent).
Butesin picrate ointment (1 per cent).	Trichophytin (full strength).
Resorcin (1 per cent).	Old tuberculin (full strength).
Quinine hydrochloride (1 per cent).	Paraphenylendiamine (2 per cent).
Sodium dichromate (1 per cent).	
Orris root (10 per cent).	

Fatty Substances

Wheat oil	Banana peel oil
Oat oil	Lemon peel oil
Rice oil	Grape fruit peel oil
Barley oil	Tea oil
Chicken fat	Timothy oil
Beef fat	Low ragweed oil
Codfish oil	House-dust oil
Coconut oil	

45. Sulzberger, M. B.; Spain, W. C.; Sammis, Florence, and Shahon, H. I.: Studies in Hypersensitiveness in Certain Dermatoses: I. Neurodermatitis (Disseminated Type), J. Allergy **3**:423, 1932.

The authors used the term disseminated neurodermatitis to designate a class of cases of dermatitis which is not due to contact and which does not show any predilection for the exposed parts. In this type, the dermatitis may occur generally, is not inclined to be vesiculated and shows a predilection for the face, neck and antecubital and popliteal surfaces. There is a tendency for the skin to be dry and thickened. The family and personal histories are usually positive for allergy of one type or another. The patient usually gives a history of infantile eczema. The patients usually show multiple sensitivity to some of the foods or inhalant allergens, and give negative reactions to patch tests almost without exception. Sulzberger and his associates emphasized the frequency with which positive reactions to silk were obtained and also positive reactions to the commonly ingested foods. Frequently, clinical testing with the foods which gave positive skin tests reproduced the patient's symptoms. Symptoms so produced often lasted for days or even weeks.

Therapy in the latter of the two types of cases discussed consisted of avoidance of cause, immunologic treatment and, finally, palliative treatment with tar salves, lotions and tar baths. The results obtained were surprisingly successful. Roentgen therapy was not used, although it is recognized as one of the best symptomatic remedies. Pilocarpine hydrochloride, from 5 to 15 drops before meals and at bed time, was recommended as worthy of trial, not only in this type of case, but in some of the nasal and bronchial allergies which do not respond to atropine, ephedrine and epinephrine.

Mucous Secretion in Asthmatic Bronchi.—Steinberg,[46] in studying the mechanism of asthmatic attacks in chronic allergy, stated that three factors are recognized as a cause of obstruction to breathing: (1) spasm of smooth muscle, which in chronic cases leads to hypertrophy of the bronchial muscles; (2) swelling of the membranes analogous to that noted in pollen hay fever; (3) bronchial occlusion due to a mucous glandular secretion.

From a study of roentgen findings after injections of iodized poppy-seed oil, 40 per cent, into the trachea during and following asthmatic attacks and from a study of autopsy material and preautopsy reports, Steinberg was inclined to believe that bronchial spasm initiates attacks, as a rule. He expressed the belief, however, that the obstruction which he found for one or several weeks following an acute attack of asthma and which he found in fatal cases is due more to the effect of tenacious mucous plugs which are found in the bronchi at autopsy and which adhere to the membranes so tightly as to be difficult to dislodge.

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46. Steinberg, Bernhard: The Mechanism of an Asthmatic Attack in Chronic Allergic Asthma, J. Allergy **3**:139 (Jan.) 1932.

These mucous plugs, which occur in the smaller bronchi, do not occur diffusely throughout the lung. They are inclined rather to involve certain areas more than others so that the roentgen findings, especially in chronic cases, indicate the involvement of a limited number instead of all the bronchi. This type of involvement, Steinberg thinks, and I believe correctly, is due to mucous plugs rather than to swollen membranes or bronchial constriction.

Peptic Ulcer and Canker Sores.—Beecher [47] reported results which led him to believe that the allergy causing local reactions, such as the Arthus phenomenon, is responsible for certain herpetic eruptions and urticarial lesions which produce a lessened resistance to the action of digestive ferments in the gastro-intestinal tract and in this way cause such conditions as canker sores and peptic or colonic ulcers. The slowness of such lesions in healing is due, he believes, to the lessened resistance of the ulcerated tissues to digestive ferments caused by the state of allergy.

Kern and Stewart [48] presented evidence which they believe tends to show that food allergy may be an etiologic factor in a small but appreciable percentage of cases of duodenal ulcer. Its recognition, they believe, puts the treatment of the patient on a sound and successful basis of removal of cause.

Epilepsy.—For a number of years, Spangler [49] has endeavored to find a possible relationship between allergy and true essential epilepsy, and he expressed the belief that a relationship actually exists which may be similar to that which exists between experimental lesions of the brain produced in animals and the administration of convulsive poisons. He referred to the work of Miller and Dandy,[50] in which experimental lesions of the cerebrum in cats did not cause epilepsy unless a convulsive poison, such as absinth, was administered. He thinks that with an epileptic foundation in human beings a convulsive effect of allergy might act in a similar way.

The chief facts which Spangler brings forth in favor of this contention lie in the finding of allergic symptoms in a large proportion of the members of epileptic families and in a large proportion of epileptic

47. Beecher, W. L.: Allergy as a Factor in Ulcers of the Digestive Tract, J. Allergy **3**:418, 1932.

48. Kern, R. A., and Stewart, S. G.: Allergy in Duodenal Ulcer: Incidence and Significance of Food Hypersensitiveness as Observed in 32 Patients, J. Allergy **3**:51 (Nov.) 1931.

49. Spangler, R. H.: Some Allergic Factors in Essential Epilepsy, J. Allergy **3**:39 (Nov.) 1931.

50. Miller and Dandy, cited by Collier, in Price, F. W.: A Textbook of the Practice of Medicine, ed. 3, New York, Oxford University Press, 1929, p. 1530.

patients, such allergic manifestations being asthma, hay fever, hives, eczema and migraine.

He found routine skin testing of little assistance in diagnosis. This, in general, is true in the case of food allergy in which there are general manifestations of reaction and should not be used as evidence against Spangler's point of view. He pointed to a similarity between idiopathic epilepsy and bronchial asthma in biochemical reactions, the clinical behavior and the results which follow therapy in allergic patients.

Spangler's points of view are not out of line with present-day ideas concerning epilepsy and allergy. He did not state that epilepsy is a direct symptom of allergy in the same sense that hay fever and asthma are direct symptoms, but expressed the opinion rather that allergy is a contributory cause of the attacks in patients who inherit an epileptic foundation. It would seem analogous in a way to the only case of locomotor ataxia which I have observed in a truly allergic patient. This patient, who had a mild beginning case of tabes dorsalis, was sensitive to peach skin. The eating of peach skin would invariably precipitate lightning pains. With the avoidance of peaches, he could reduce the number of attacks.

Calcium, Phosphorus and Potassium Studies.—Lierle and Sage [51] studied the calcium, phosphorus and potassium content of the serum and spinal fluid in patients with asthma. The results obtained do not show any marked difference from accepted normal values.

Sinusitis.—Kern and Donnelly,[52] from a clinical and roentgenologic study of patients with hay fever and seasonal asthma, showed that the incidence of sinus trouble is not grossly different from that of normal people in purely seasonal cases. In perennial cases, however, there was a higher incidence of sinus disease.

Skin tests and the results of specific treatment supported their view that the asthma complicating hay fever is in the gross majority of cases due to hypersensitiveness to pollen or other inhalant substances and not to sinus disease. They expressed the belief that sinus disease, in the majority of cases, is a secondary complication and not a primary factor in the case of asthma.

Enuresis.—Bray [53] discussed enuresis of allergic origin. He stated that children with the complaint often have their worst symptoms coincident with other manifestations of allergy, and if the offending allergens are removed, the symptoms in the bladder are less severe or

51. Lierle, D. M., and Sage, R. A.: A Study of the Calcium, Phosphorus and Potassium of the Serum and Spinal Fluid in Asthma, J. Allergy **3**:325, 1932.

52. Kern, R. A., and Donnelly, J. C.: The Incidence and Rôle of Paranasal Sinus Disease in Hay Fever, J. Allergy **3**:172, 1932.

53. Bray, G. W.: Enuresis of Allergic Origin, Arch. Dis. Childhood **6**:251, 1931.

disappear. He compared the contraction of the bladder musculature with the contraction of the bronchial musculature in asthma and expressed the opinion that the two illnesses may have a similar origin in certain types of cases.

Psoriasis.—Zeidler [54] brought up the question as to whether or not psoriasis is an allergic disease. This question has been asked, and, so far as I know, has remained unsolved for a period of at least fifteen years. It is a question that is worthy of thorough study. Zeidler did not answer the question, but stated that in a series of patients treated with pollen extract for hay fever, several persons who suffered from psoriasis became symptom-free. He tested several patients who had psoriasis and obtained positive skin reactions in each case with one material or another, and found further that 22 per cent of the patients who had psoriasis also showed other symptoms typical of allergy.

Eosinophilia.—J. Adam [55] stated that eosinophilia characterizes most attacks of asthma. An eosinophilia so produced can be reduced with epinephrine, or atropine or by a cold bath, the latter, he believes, because of the stimulation of the suprarenals. Fever reduces the eosinophil count, likewise, he believes, through stimulating the suprarenals.

Arthritis.—Harkavy and Hebald [56] reported that in a study of 400 adults with asthma, 9 patients had attacks of arthritis which came to the fore during asthmatic attacks. They attributed the arthritis to a complicating infection of the bronchial tree and expressed the belief that whereas the asthma may have been due primarily to some inhalant substance, the manifestations of arthritis were not associated with this but instead with the complicating infection.

TREATMENT

Specific Treatment.—Brown [57] reported further results on the perennial treatment of hay fever, stating that the method gives results which are as good or better than preseasonal treatment and apparently are more permanent. He stated that the method is easier on both physician and patient. Treatment is started at any time of the year, preferably several months before the season commences, is worked up to an optimum dose for the patient and is continued at this level indefinitely at approximately monthly intervals.

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54. Zeidler, R.: Is Psoriasis an Allergic Disease? Wien. med. Wchnschr. **81**: 877, 1931.

55. Adam, J.: Report on Asthma Research at Stobhill Hospital, Glasgow, Glasgow M. J. **116**:81, 1931.

56. Harkavy, Joseph, and Hebald, Selian: Association of Infectious Asthma and Arthritis from the Point of View of Bacterial Allergy, J. Allergy **3**:87, 1931.

57. Brown, Aaron: The Perennial Treatment of Seasonal Hay Fever, J. Allergy **3**:113 (Jan.) 1932.

Brown [58] reported on the treatment of pollen hay fever with doses of extract which are somewhat larger than the average employed by other observers. In his preseasonal treatment, he administered up to 1 cc. of a 6 to 10 per cent extract of pollen, and stated that with this increased dosage he had eliminated the small percentage of failures following the treatment by the methods formerly used.

According to the results of Carter, Black, Piness and others, it is questionable whether or not a concentration of pollen atopens greater than that found in a 3 per cent extract can be obtained. In other words, they believe that a 3 per cent extract of pollen saturates the extracting fluid. If this is so, Brown's method of administration at least assures a saturated solution. His 1 cc. dose is a little greater than that used by other observers.

In the middle west, 5 per cent extracts have been used for many years, the dose being pushed up to a little above 0.5 cc., and this has been found necessary for the obtaining of desirable results in a gross percentage of pollen cases. It is surprising to find that in Brown's district, where the concentration of pollen in the air is so much less than in the middle west, smaller doses do not suffice. However, it is now known that the symptomatology of the patient with hay fever is not dependent entirely on the pollen concentration of the air and that many persons have symptoms when pollen counts in their district are far below the maximum.

The experience of a great number of workers in this field harmonizes with Brown's in the finding that large doses of extract are necessary for successful results in most pollen districts.

Colmes,[59] comparing the positiveness of skin tests before and after specific pollen therapy, found that in 9 per cent of cases, the skin tests remained positive and the disease was but little influenced by pollen therapy. In 91 per cent of patients, however, the skin tests had a tendency to diminish and corresponded with clinical results with such constancy that he drew the conclusion that there was a definite parallelism between the degree of dermal reaction and the state of the patient's sensitiveness to pollen. This parallelism, however, applies only to tests made at different times in a given person; for he found that no test was expressive of a given degree of clinical sensitiveness for all patients.

Colmes expressed the belief that repeated dermal tests can be used as a guide in pollen therapy as to the change in the state of a persons'

58. Brown, G. T.: Maximum Dosage in Pollen Therapy, J. Allergy **3**:180, 1932.

59. Colmes, Abraham: Serial Skin Tests as a Guide in the Treatment of Hay Fever, Preliminary Report, J. Allergy **3**:449, 1932.

clinical sensitivity. He expressed the opinion that therapy should be pushed to the point of neutralization of the dermal reaction.

Rowe [60] discussed desensitization in the case of food allergy. He stated that a determination of the allergy-producing foods by diet and by the skin reaction is most important and that in the case of multiple sensitization it might be quite difficult. Several methods of desensitization are recommended. First, the offending foods are eliminated from the diet. When this was complete and continued for a long period of time, Rowe found that a certain proportion of patients, especially infants, cease to react cutaneously to the foods that originally caused marked positive cutaneous reactions.

Oral desensitization, which was previously discussed by Schloss,[61] Walker [62] and others, is also recommended as successful in a certain proportion of cases. All observers agree in recommending the complete avoidance of the offending food followed by its administration in the beginning with infinitesimal quantities of the food. The amount can be increased by degrees until considerable quantities can be given without the production of symptoms that originally followed the ingestion of unbelievably small quantities of the offending food. Rowe found that this therapy, if given successfully, may result in positive skin reactions becoming negative. Rowe was less enthusiastic about the results obtained by hypodermic desensitization. This method of therapy has been disappointing to many students of this line of work, although strikingly successful in a small proportion of cases. Rowe stated that even this therapy should be followed by immunization treatment by mouth. In discussing peptone therapy, he stated that he has discontinued intravenous injections in his work because it occasionally causes shock. In only 1 of 4 patients treated with peptone were the skin tests definitely reduced. He stated that his observations on the effect of specific proteins on the various manifestations of food allergy admit no definite conclusions. He is not enthusiastic about the denaturization of foods to make them available for susceptible patients. The use of calcium, quartz light, vitamin therapy, protein therapy and autohemotherapy was found to be of no definite value in the treatment of patients sensitive to food.

Effect of Surgical Intervention and Anesthetics on Asthma.—Vallery-Radot, Blamoutier and Claude [63] reported a study of 15 cases

60. Rowe, A. H.: Desensitization to Foods, J. Allergy **3**:69 (Nov.) 1931.

61. Schloss, O. M.: A Case of Allergy to Common Foods, Am. J. Dis. Child. **3**:341 (June) 1912.

62. Walker, I. C.: The Treatment of Patients with Bronchial Asthma with Subcutaneous Injections of the Proteins to Which They Are Sensitive, J. M. Research **36**:423, 1917.

63. Vallery-Radot, P.; Blamoutier, P., and Claude, F.: The Influence of Surgical Intervention on Asthmatic Crises, Bull. et mém. Soc. méd. d. hôp. de Paris **47**:416, 1931.

of asthma in patients who were relieved for periods varying from two weeks to six months by a surgical operation even though the anesthetic used was local. They did not think that the relief could be attributed in all cases to the anesthetic used. They also reported 6 cases of nasal allergy which were complicated by asthmatic crises following surgical intervention. They stated that it is conceivable that surgical procedures and anesthetics can modify the state of tone in the vegetative nervous system and might either excite or stop crises of this sort. Regardless of explanation, however, they stated that surgical intervention can have either effect.

Weille [64] selected 40 patients for nasal surgical intervention from a group of several hundred asthmatic patients that he examined. In all of these, one or a number of the sinuses were definitely proved to be diseased.

Of this group of 40 cases, 75 per cent showed a favorable result in the nose following operation, but only about 50 per cent showed long-continued, favorable changes in their asthmatic symptoms.

Of the 6 patients having extrinsic asthma, none was improved.

Weille stated that the most favorable cases for surgical improvement are in patients with asthma associated with polypi in the sinuses, and not in patients with thickened membranes or cysts.

The indications for operation, he considers, are about as follows: pathologic changes in the sinuses demanding surgical treatment on their own merit; recurrent head colds precipitating attacks of asthma; a downward vicious circle in patients with severe cases of asthma, which can sometimes be broken by temporary relief, and indications that operation may be helpful, as shown by improvement after sinus irrigation.

Troisier and Boquien [65] reported a study of 3 cases of asthma in patients who were relieved by general anesthesia; in 1 case the anesthesia was given for operative purposes and in 2, directly to test the possible efficacy of general anesthesia in the treatment of chronic severe asthma. The authors referred to the observations which have been made frequently, that asthmatic patients frequently are improved or actually become asthma-free following operation and sometimes remain asthma-free for prolonged periods of time. The authors, in their own cases in which the patients were relieved in this way, were unwilling to attribute the improvement to the operation itself, but rather to the general anesthesia. In each of the 3 cases of chronic asthma in which anesthesia was applied, they obtained marked relief, which they attributed directly to the effect of the anesthetic.

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64. Weille, F. L.: Studies in Asthma: XVIII. The Surgical Treatment of Chronic Sinusitis in Asthma, J. Allergy **3**:518, 1932.

65. Troisier, J., and Boquien, Y.: Treatment of Asthma by General Anesthesia, Bull. et mém. Soc. méd. d hôp. de Paris **47**:310, 1931.

The giving of a general anesthetic to a patient with asthma is a bold procedure. In spite of this, further observations and efforts to relieve patients with severe asthma in this way are not only justified but indicated. There is a type of asthma which is extremely severe and in which a vicious circle is established, caused by the fact that the patient cannot either eat or sleep, and which can often be broken by adequate rest.

Bronchoscopic Treatment.—Weille [66] reported the effect of bronchoscopy on the symptoms of asthma. Of 15 patients so treated, 4 received no benefit. He stated that 1 patient described himself as cured when heard from nineteen months after treatment. One was practically cured a year after treatment. A third was cured for a period of fifteen months with the exception of a single attack. In a fourth patient, the procedure was spoken of as life-saving. In the remaining 7 per cent, there was marked relief which lasted for from two weeks to seven months following each bronchoscopic treatment. Weille stated that the most typical single pathologic finding in the cases was the presence of a thick, viscid, tenacious secretion in the bronchial tree. The character of the secretion was considered more important than the quantity, since when the mucus is viscid enough, even a small quantity that cannot be dislodged by coughing can grossly obstruct several large bronchi. The patients who had no bronchial secretion were not benefited by bronchoscopy.

If the foregoing findings are compared with those of surgical intervention in general in relationship to asthma, some explanation of the aforementioned benefits might be obtainable. It is certainly beyond the realm of imagination to think that the removal of a few flecks of mucus such as could be reached by a bronchoscope could by any possible chance have more than a short temporary effect on the course of asthma. However, a local anesthetic combined with the effect of having metal tubes introduced into the trachea might have an analogous effect to that referred to by Vallery-Radot in relationship to surgical procedures in cases of asthma. The trachea and bronchial tree, like the urethra, are not accustomed to contact with foreign bodies as are the mucous membranes of the trachea and the rectum, so that the introduction of a metal tube into either the trachea or the urethra has an effect on the vegetative nervous system which is far greater than that which would be experienced following the use of an esophagoscope or a proctoscope. To me, it is conceivable that the upset to the autonomic nervous system combined with a local anesthetic used in bronchoscopy might affect the patient in a manner analogous to that of a surgical operation. I can

66. Weille, F. L.: Studies in Asthma: XIV. Bronchoscopy in Asthma, J. Allergy **3**:206, 1932.

conceive of a catheter chill breaking an attack of asthma. I should think that bronchoscopy might make some patients better and others worse. The recommendation of bronchoscopy for lasting benefit in the treatment of asthma seems anything but logical. However, in the treatment of allergy one is dealing with unknown factors, and it is possible that something, at present unknown, might be reached by bronchoscopy which might affect the course of asthma in one way or another.

Kern, in his discussion of this article, does not hesitate to say that he has seen amazing and alarming reactions follow the use of iodized oil in patients with asthma, and that such material, if injected during bronchoscopy, ought to be removed as soon as possible.

Fever Artificially Induced.—It is recognized by all students of allergy that fever, almost regardless of cause, may relieve allergic disease for days, weeks, months or years. This peculiar circumstance has been the subject of much discussion and has not as yet been satisfactorily explained. I [67] observed many years ago that heat-sensitized patients presenting symptoms such as hay fever, asthma, urticaria, dermatitis or what not reacted most markedly to heat when the body temperature was lowest, and that the symptoms, whatever they might have been, disappeared with fever and could be relieved by the artificial production of fever with injections of Bacillus coli. I ventured the suggestion that the relief of asthma which may follow infection, even bronchopneumonia, might be due to the fact that heat-sensitive patients are relieved by fever almost regardless of its source. I frankly admit, however, that it takes more than this to account satisfactorily for the gross relief which is likely to be obtained in a vast number of patients with chronic allergy by the symptom of fever. Rackemann [68] stated that asthma, hay fever, chronic vasomotor rhinitis, urticaria and infantile eczema clear up when fever occurs from any cause. Berg [69] reported the cases of 2 patients with asthma who were relieved after an attack of lobar pneumonia. Holst [70] stated that while asthmatic patients may be symptom-free during febrile attacks, the relief thus obtained is rarely permanent.

Feinberg, Osborne and Afremow,[71] on the basis of results such as those mentioned and on the basis of the fact that varied types of fever-

67. Duke, W. W.: Heat and Effort Sensitiveness, Cold Sensitiveness, Arch. Int. Med. **45**:206 (Feb.) 1930.

68. Rackemann, F. M.: Clinical Allergy, New York, The Macmillan Company, 1931, p. 484.

69. Berg, S.: Relief of Bronchial Asthma Following Lobar Pneumonia, J. Allergy **2**:54, 1930.

70. Holst, P. F.: Fever in Relation to Asthma, Norsk mag. f. lægevidensk. **83**:577 (Aug.) 1922.

71. Feinberg, S. M.; Osborne, S. L., and Afremow, M. L.: Fever by Diathermy in the Treatment of Allergic Disease, J. Allergy **2**:414, 1931.

producing substances given hypodermically may relieve asthma temporarily, studied the effect on asthmatic symptoms of fever produced artificially through the agency of diathermy. It must be mentioned here, on account of its practical importance, that the authors referred to the dangers to the patient with allergy of fever-producing substances, even of the effect of hot baths. I have repeatedly warned against the careless use of heat of any sort in patients with asthma, urticaria or dermatitis who prove to be sensitive to heat. In my early work on patients sensitive to heat and effort, I frequently precipitated reactions which were all but fatal by amounts of heat such as with exposure of the arm to the heat of a nitrogen lamp for a few seconds or to a cloth wet with water at 42 C. for a few seconds and might, in several instances, have precipitated a fatal attack had I not had ice handy for rapid use in stopping a reaction so produced.

Being aware of the dangers of this form of therapy in certain types of cases, Feinberg and his associates carefully studied the effect of diathermy in the treatment of allergy and summarized their work as follows.

There is no doubt that the treatment of fever by diathermy as here described is capable of giving benefit to some sufferers from asthma. The disadvantages of this form of treatment are the expense, the discomfort to the patient, the time consumed by the operator, the temporary nature of the relief and the incomplete knowledge of the limitations of its uses. Further disadvantages lie in the necessary use of caution and the selection of cases suitable for this treatment and the strenuous burden it may place on the patient. Feinberg and his co-workers stated that it is necessary to make careful preliminary studies before this treatment is undertaken, and they think that at best the treatment should be restricted practically to those who have resisted other forms of therapy. They stated that the benefit to the patient, when some is obtained, does not depend on any decided change in the reagins of the blood.

Leopold and Stewart [72] reported a study of the relationship between fever and asthma and found, in harmony with general belief, that fever of varying sources gives temporary relief. However, the authors expressed the belief that in the case of asthma in which the patient is relieved by infection, fever cannot be looked on as the only factor.

Miller and Piness [73] studied the effect of fever produced by a high frequency electric current on 7 cases of asthma which were not com-

72. Leopold, S. S., and Stewart, S. G.: The Effects of Fever, Either Accidentally Incurred or Artificially Produced, in Bronchial Asthma, J. Allergy **2**:425, 1931.

73. Miller, Hyman, and Piness, George: Hyperthermia Induced by High Frequency Electric Current in the Treatment of Intractable Asthma, J. Allergy **2**:436, 1931.

plicated by other diseases. They did not draw conclusions, but could not report on this as a promising method of treatment.

PHYSICAL ALLERGY

There has been considerable discussion in the literature on the physical allergies caused by such agents as heat, cold, light and scratches. An attempt has been made to find a relationship between the manifestations of this type of illness and the reactions caused by histamine, especially by Lewis,[74] Horton and Brown,[75] Alexander [76] and others.

A study of this type of reaction has been grossly neglected by students of allergy. I cannot understand this, because reactions of this sort are grossly important whether the illness is directly related to true atopy or not. Certainly, the clinical manifestations are identical even though the pathogenesis may be different. These illnesses, just as drug allergies, have to be taken into consideration by students of the subject.

In my earlier work on this subject,[77] I was inclined to believe that certain types of physical allergy might be due to a liberation of histamine in the tissues by a specific physical agent. Lewis,[74] working independently and at the same time, was more energetic in his endeavors to prove this point definitely. However, the technical difficulties of either proving or disproving the idea are gross, and at the present time one has to base one's opinions, pro or con, on indirect evidence. In spite of the fact that local reactions caused at the site of contact with physical agents resemble reactions to histamine, this is not the common type of illness which follows exposures in the case of sensitiveness to heat and effort and to cold. In the latter type of illness, the reaction is caused by an increase in the sense of heat or cold and appears in distant structures, even when heat or cold is applied distal to a tourniquet around the arm, which stops the circulation of the blood and which would prevent the dissemination of histamine if formed in the tissues which react. Whereas Horton and Brown have found cases of urticaria caused by cold in which the reaction occurred on release of the tourniquet, these are the exception instead of the rule. In the gross majority of cases, symptoms appear within a few seconds of the time of exposure and prior to the release of the tourniquet.

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74. Lewis, Thomas: Supplementary Notes upon Reactions of Vessels of Human Skin to Cold, Heart **15**:351, 1931.

75. Horton, B. T., and Brown, G. E.: Systemic Histamine-Like Reactions in Allergy Due to Cold: Report of Six Cases, Am. J. M. Sc. **178**:191, 1929.

76. Alexander, H. L.: "Physical Allergy": Report of a Case with Successful Treatment, J. Allergy **2**:164, 1931.

77. Duke, W. W.: Urticaria Caused Specifically by the Action of Physical Agents: (Light, Cold, Heat, Freezing, Burns, Mechanical Irritation, and Physical and Mental Exertion), J. A. M. A. **83**:3 (July 5) 1924.

Heat Sensitiveness.—Wilson [78] reported a case of sensitization to heat in which, during the summer months, flushing of the skin, urticarial wheals and itching developed whenever the patient worked and became a little too warm. These changes would subside when the patient cooled off. He was given thyroid extract, ⅓ grain (0.02 Gm.), three times daily, with gross clinical improvement.

Wilson classified the case as one of physical allergy caused by sensitiveness to heat.

Light Sensitiveness.—Weiss [79] reported a case of urticaria solaris (urticaria caused by light) which is interesting in that adequate exposure of a sufficient amount of skin to light would cause urticarial lesions not only at the site of contact with light, but in distant structures as well, such as on the penis and on the feet. He referred to cases previously reported by Duke,[80] Frei,[81] Beinhauer,[82] Vallery-Radot and his associates,[83] Wucherpfennig [84] and Sellei.[85] Beinhauer reported 3 cases and made an exhaustive study as to which rays of light were responsible for the reaction. In 1 of his cases, all but the red rays of light would give rise to a similar pathologic process in the skin.

From the number of cases now reported, this type of allergy must be looked on as definitely proved and due directly and only to sunlight in certain cases. It can apparently be caused by different rays of light in different patients and occasionally, as mentioned in Beinhauer's case, by a wide variety of rays of light. In the case which I reported, the patient was more affected by rays in the blue end of the spectrum than by ultraviolet light. In the cases reported by other observers, the actinic ray seems to have been the more offensive.

No explanation of the mechanism of this aype of sensitiveness has as yet been proved. Weiss, however, mentioned that the majority of cases occur in women and that in Beinhauer's 3 cases the affliction seemed to be associated with a disturbance in menstruation.

78. Wilson, R. A.: Heat Sensitiveness, J. Allergy **2**:499, 1931.

79. Weiss, Edward: Urticaria Solaris, J. Allergy **3**:192, 1932.

80. Duke, W. W.: Urticaria Caused by Light, J. A. M. A. **80**:1835 (June 23) 1923.

81. Frei, W.: Local Urticarial Skin Reactions to Sunlight, Arch. f. Dermat. u. Syph. **149**:124, 1925.

82. Beinhauer, L. G.: Urticaria Solaris: Report of a Case, Arch. Dermat. & Syph. **12**:62 (July) 1925.

83. Vallery-Radot, P.; Blamoutier, P.; Besançon, and Saidman: Urticaria from Sun's Rays, Bull. et mém. Soc. méd. d. hôp. de Paris **50**:1116, 1926; Urticaire solaire (presentation de la malade), ibid. **50**:1122, 1926.

84. Wucherpfennig, V.: Pathologische Lichtüberempfindlichkeit in qualitativer und quantitativer Hinsicht, nebst Untersuchungen zur Pathogenese der Lichtquaddel, Arch. f. Dermat. u. Syph. **156**:520, 1928.

85. Sellei, J.: Beiträge zu den physikalischen Allergien: Prurigo solaris (prurigo aestivalis seu prurigo simplex chr. recidivans) und Eczema solare, Arch. f. Dermat. u. Syph. **161**:32, 1930.

The similarity of a light reaction of this type and a histamine reaction has been noted by almost all who have studied this problem. Furthermore, a similarity between this illness and the contact reaction caused by cold and the type caused by scratches (formerly called urticaria factitia) has been noted by all observers. Furthermore, a similarity between light sensitiveness caused by hematoporphyrinemia in human beings and that observed in white animals after the eating of buckwheat has been noted with interest. In spite of all this, however, the exact mechanism through which this reaction takes place has been explained only through inference or indirect experiment. Especially interesting are the cases of Thomas Lewis and his associates,[86] in which the reactions are shown to be similar in so many respects to histamine reactions. On account of the difficulty or impossibility of passive transfer of this type of sensitiveness, I am inclined to class the reactions with the drug allergies rather than with atopy. Thomas Lewis alone has been able to find evidence of passive transfer, and only in the case of sensitiveness to cold. A possible relationship between this type of illness and vernal catarrh has been recognized by Ruedemann, and successful therapeutic results were reported in the treatment of vernal catarrh through exposing the skin to increasing quantities of light.

Cold Sensitiveness.—Bray[87] reported the case of a patient with physical allergy caused by cold in which the immersion of the hands in cold water caused localized itching and swelling after a few minutes and, finally, brawny induration to such an extent that the hands would be twice their normal size. The patient was then unable to flex the fingers. Following this, he would have an irritating cough and symptoms referred to as a cold.

I[88] discussed the treatment of physical allergy, that is, of heat and effort sensitiveness and cold sensitiveness, and the treatment of contact urticaria caused by light, cold and scratches. Treatment of these conditions is reasonably successful in the gross majority of cases and brilliantly successful in selected cases. Treatment is based on the use of the same principles which are employed in the treatment of allergy caused by material agents, that is, by avoidance of the cause and by increasing the tolerance for the noxious agent by graduated increasing exposures to the agent responsible for the illness.

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Abstracts from Current Literature

Ear

INFLUENCE OF SYMPATHETIC NERVOUS SYSTEM ON VESTIBULAR EXCITABILITY. G. CANTELE AND K. GRAHE, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**:281 (Feb. 25) 1932.

Cantele and Grahe state that vestibular examinations performed before and after division of the cervical sympathetic nerve and during stimulation of the peripheral sympathetic stump show that stimulation and paralysis of the sympathetic cervical nerve influence vestibular excitability. They found that division of the cervical sympathetic nerve effects a pronounced decrease in frequency of the nystagmus provoked by cold irrigation on the side operated on. The duration of the cold nystagmus on the side operated on is regularly decreased; the latent period is slightly increased. In hot irrigation the opposite type of reaction can be detected. Rotatory stimulation shows a general decrease of excitability without favoring one side. On the same side, there is a barely perceptible decrease in duration of the nystagmus and of the latent period. Hot irrigation during stimulation of the sympathetic nerve does not show a uniform reaction type. The authors think that the investigations are additional proof that the vasomotor component of caloric stimulation only has a modifying action on the actual vestibular excitability.

EDITOR'S ABSTRACT.

ROENTGENOLOGIC AND CLINICAL INVESTIGATIONS OF SECONDARY SCLEROSIS OF MASTOID PROCESS. W. MEYER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**:292 (Feb. 25) 1932.

Meyer reports the results of a roentgenologic investigation aiming at an answer to the much debated question of secondary sclerosis of the mastoid process. Twenty-eight patients in whom a well pneumatized mastoid process had been roentgenologically determined at the onset of acute otitis media were reexamined during the course of disease and several months later. In twenty-four of the cases there were no roentgenologically demonstrable changes in the mastoid process following the acute otitis media. In four cases a partial substitution of new growth of bone for pneumatized areas was seen after the otitis. However, a complete secondary sclerosis of a pneumatized mastoid process was not observed in any of the cases. The four cases in which a partial obliteration of previously existing cells occurred were exceptionally severe. In all four cases signs of erosion of the bony cell outlines were observed, sometimes at an early stage of suppuration. This confirms Wittmaack's statement that severe inflammations which destroy the epithelial covering of the cells, thereby inducing the growth of connective tissue or secondary formation of new bone, must also cause destruction of the adjacent thin trabeculae of bone. A complete secondary sclerosis of the cell system would be extremely rare, if it occurs at all, as it would have to be preceded by a total destruction of the cell system; such an extensive destructive process would usually lead to vital complications or operation. That there may, however, be spontaneous healing with partial secondary sclerosis of the cells is shown by the author's cases. Krainz' assumption that an unnoticed sclerosis occurs through latent mastoiditis finds no support in these results. The partial secondary sclerosis demonstrated in a few cases shows that in rare cases of poorly pneumatized mastoid processes with certain anamnesis of mastoiditis or severe otitis media a partial secondary obliteration of the cell system through inflammation may be assumed in addition to the primary disturbances of development assumed by Wittmaack. This supports rather than contradicts the latter's theory of the origin of a slightly, or not at all, pneumatized mastoid process.

EDITOR'S ABSTRACT.

INVESTIGATIONS WITH OTO-AUDION ON DEAF-MUTES. K. GRAHE, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**:302 (Feb. 25) 1932.

Grahe reports the results of investigations comparing the hearing for speech with the hearing for tone. The capacity for tone hearing was determined by means of the oto-audion, an instrument which permits the electrical production of every audible tone, with forty-five gradations of intensity for each tone, each being one and eighty-three hundredths times stronger than the preceding one. The tones are transmitted through a loud speaker. In the examination of totally deaf children with the oto-audion, a feeling curve of definite character can occur. By use of a telephone for bone conduction only, a feeling curve can be determined even in deaf mutes with a slight capacity for hearing. In the case of deaf mutes who hear through air conduction, the hearing curves for unintensified and intensified tones of the oto-audion are parallel only in their general outline. The extension of the hearing curve is essentially dependent on the intensity of the test tone. With high intensity the tone gaps disappear. The less the hearing capacity of a child, the greater is the concentric restriction of the hearing for tone. The hearing capacity for speech sounds is essentially dependent on the sensitivity of hearing rather than on the quality of hearing. The author thinks that in children who, with a sufficient capacity for hearing tone, are able to perceive speech sounds through the ear, improvement of speech-hearing and pronunciation is possible by the aid of hearing tubes for instruction through the ear and control of the self-produced sounds. The only agreement between hearing for the tuning fork and for the oto-audion is that in cases in which tuning forks are heard quantitatively, a relatively large hearing for tone can be determined by intensified oto-audion tones. The author thinks that the results of the investigation show that Bezold's contention, that for speech-hearing only a certain quantitative hearing of the tone range b^1-g^2 is necessary, is untenable.

EDITOR'S ABSTRACT.

OTITIS IN NURSLINGS AND ITS COMPLICATIONS. M. KRASSNIG, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**:335 (Feb. 25) 1932.

Krassnig discusses otitis in nurslings and its complications on the basis of otologic examination of 920 nurslings, on 52 of whom autopsies were performed. The author recognizes the frequent existence of a latent otitis, by which he means a condition in which otologic examination shows a diseased tympanum but there are no subjective symptoms such as fever or pain. Otitis of this latent type, or with slight clinical symptoms, when associated with dyspepsia in nurslings, is usually due to a decreased general resistance resulting from the intestinal disturbance. This view is supported by the fact that the dyspeptic symptoms usually precede the otitic symptoms by weeks or months, and that an improvement in the general condition never results from treatment of the ear, whereas the otitic symptoms usually disappear when the intestinal function returns to normal. A decline in general health cannot be attributed to latent mastoiditis, as a suppuration of the mastoid antrum in a nursling would rapidly become noticeable at the surface or spread inwardly and give rise to endocranial complications. Of the 4 cases of endocranial complications in which the possibility of otogenous origin was admitted, clinical and sometimes autopsy findings proved this supposition wrong. The author thinks that the otitis of nurslings is not as dangerous as some otologists think or as insignificant as some pediatricians believe. Usually it is only one manifestation of a general disturbance of the organism. Diagnostic paracentesis often helps to determine the clinical importance of the otitis. If there is no secretion following paracentesis or if it stops after a short time and all other local symptoms such as inflammation of the lymph nodes, periostitis and changes in the auditory meatus are lacking, the otitis probably does not play an important rôle in the clinical picture.

EDITOR'S ABSTRACT.

ROENTGENOLOGIC EXAMINATIONS IN GRADENIGO'S SYNDROME. L. KRAUS, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**:349 (Feb. 25) 1932.

Kraus reports the roentgenologic findings in four cases of Gradenigo's syndrome, in two of which there were acute otitis and mastoiditis, and in the other two, a chronic mesotympanic disease with acute exacerbation. A disease of the apex of the pyramid, empyema, abscess of the bone and areas of lesser density could be roentgenologically determined with certainty in three of the four cases. The least involvement was a decrease in density with definitely visible contours; the most severe involvement was a real abscess of the bone with partial defects or complete absence of contours and destruction of the contours of the bone in the center. In serial roentgenograms, distinct healing tendencies of these foci in the apex of the petrous bone were visible. At first, the contours appear to become more distinct and then new growth of bone appears to take place at the edges of the abscess. The healing requires months. There is as yet no uniform conception of Gradenigo's syndrome; it appears that several processes are responsible. The roentgenographic findings discussed are not always pathognomonic for Gradenigo's syndrome. They must be properly correlated with the clinical symptoms and the time of their occurrence. The syndrome almost always occurs with an acute inflammation or with exacerbation of a chronic inflammation, especially chronic mesotympanic inflammation. In the opinion of the author, the most important result of his investigations is not the roentgenologic determination of the foci in the tip of the pyramid of the petrosa, but evidence of the healing of these foci.

EDITOR'S ABSTRACT.

CLINICAL SIGNIFICANCE OF DEPRESSION OF SHRAPNELL'S MEMBRANE. M. SCHWARZ, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:16 (April 25) 1932.

Schwarz thinks that the depression of Shrapnell's membrane is of diagnostic value. He differentiates between two types of depression, one in which the membrane is movable and is not grown to the structures below it and the other in which it has grown to its substratum and shows, at most, a minimal mobility of some circumscribed spot. The two types can be easily differentiated in clinical examination by the fact that the former can be equalized by catheterization of the tube or the use of a speculum with an air syringe while the latter remains refractory. This type represents an irreparable condition and is of serious significance in contrast to the former type which is usually associated only with transitory disturbances of tubal ventilation. On the basis of thorough otorhinolaryngologic examination of 126 patients with irreparable depression of Shrapnell's membrane, which included roentgenologic visualization of the mastoid process, and serial histologic examination of 220 temporal bones of adults and 200 of fetuses or newly born infants as well as a study of the literature, the author thinks that the irreparable depression of Shrapnell's membrane is of diagnostic value despite the variable anatomic relation between the membrane and its surroundings. It points to a chronic process of changing degree in the middle ear based on a deficient anlage of the mucous membrane of the middle ear and the concomitant predisposition to inflammatory, especially chronic inflammatory, disease. For this reason these cases always exhibit a marked disturbance of pneumatization and usually a compact mastoid process.

EDITOR'S ABSTRACT.

ACUTE MASTOIDITIS AND SUBOCCIPITAL TUBERCULOSIS. G. ENGELHARDT, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:160 (May 23) 1932.

Engelhardt reports a case of suboccipital tuberculosis in which the symptoms of the initial stage of this disease (rigid median position of the neck and occipital neuralgia) appeared during the course of an acute mastoiditis, and it was not until the ear operated on was practically healed that the appearance of gravitation abscesses in a typical location and the spontaneous rupture of a retropharyngeal abscess determined the diagnosis of suboccipital tuberculosis. Swelling at the nape

of the neck, pressure sensitivity in the median line and radiating pains in the ears, typical symptoms of suboccipital tuberculosis, were lacking in this case. The author thinks that the tuberculosis of the atlas (disease of the atloido-occipital joint was not roentgenologically demonstrable) existing in latent form was brought to an acute exacerbation by the operative trauma and postoperative inflammation of the lung.

EDITOR'S ABSTRACT.

Pharynx

END RESULTS IN TONSILLECTOMY BY ELECTROCOAGULATION DUE TO IMPROVED TECHNIC. J. A. HAIMAN, M. J. & Rec. **135**:471 (May 18) 1932.

The electrocoagulation of tonsils with the bipolar tonsil electrode is under the definite control of the operator and therefore may be regarded as a true, scientific procedure. Constant duplication of end-results can be obtained, with the elimination of heretofore existent dangers of coagulating the peritonsillar vessels. Bipolar electrocoagulation permits the removal of all of the tonsillar tissue and affords end-results comparable to those of the orthodox surgical procedure. Tonsillectomy by electrocoagulation is a valuable contribution to rhinolaryngologic surgery.

WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

LOCAL ANAESTHESIA FOR TONSILLECTOMY. J. W. COSTELLO, Brit. M. J. **1**:839 (May 7) 1932.

The author, an advocate of local anesthesia in tonsillectomy, describes his experience in a small series of cases.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

POSTTONSILLITIS GRAVITATION ABSCESSES. W. MAYER, Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **131**:125 (April 25) 1932.

Meyer reports a case of a circumscribed gravitation abscess originating in the peritonsillar region and descending into the mediastinum which healed without surgical intervention other than incision of the peritonsillar abscess from which the suppuration spread. In this case, eleven days after incision of a large peritonsillar abscess, occurring in connection with tonsillitis, the patient vomited a large amount of green pus. Roentgenologic visualization of the abscess cavity by injection of an opaque fluid showed that suppuration descended into the spatium retropharyngeum and the posterior portion of the mediastinum between the esophagus and the spinal column, ending in the thorax at the level of the fourth thoracic vertebra. As esophagoscopy showed that there was a rather broad strip of apparently healthy mediastinal connective tissue between the abscess and the esophagus, drainage by division of the esophagus was not undertaken. With rest in bed and elevation of the pelvis the patient improved rapidly. Roentgenoscopy after six days showed a completely normal thorax; the lungs and mediastinum were clear, and no trace of the opaque medium remained. Visualization with a contrast fluid may be recommended as a diagnostic aid. The achievement of complete cure by conservative therapy should lead to careful evaluation of surgical successes in similar cases. The necessity of differentiating between a circumscribed gravitation abscess and the much more frequent phlegmons in the throat with general septic symptoms is stressed.

EDITOR'S ABSTRACT.

SYMPTOM OF COMPLICATION FOLLOWING ADENOTOMY. H. SCHAEFER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:231 (May 23) 1932.

Schaefer reports a symptom of complication after adenotomy which he has observed frequently of late but of which he finds no mention in the literature. It consists in severe circumscribed pain at the nape of the neck, setting in soon

after the operation and impeding certain movements of the head. The manifestation usually recedes within a week. There is no pressure sensitivity in the affected part, the lymph nodes at the nape of the neck are not palpable and other cervical lymph nodes are the same size and consistency as before the operation. The possible theories for the development of this symptom (mechanical, inflammatory and infectious) are critically considered. The most probable explanation in the opinion of the author is that the pain is a symptom of a lymphangitis, or lymphadenitis, resulting from surgical opening of lymph channels and consequent spread of infected lymph thrombi.

EDITOR'S ABSTRACT.

THE FREQUENT APPEARANCE OF ENCEPHALITIS AFTER NONSPECIFIC TONSILLITIS. FANNY HALPERN, Jahrb. f. Psychiat. u. Neurol. **48**:154, 1932.

In the Vienna University Clinic for Psychiatry and Neurology the author observed six cases of encephalitis between the early part of September and the middle of December, 1931. The subjects were all women in whom the disease was a complication of tonsillitis. The encephalitic manifestations appeared early in the course of the sore throat. It is noteworthy that the encephalitis was in every case unusually benign. Thus far no fatality has occurred; four of the patients are on the road to recovery, and four have completely recovered. Obviously, the further fate of these patients cannot as yet be foretold. It is also noteworthy that the neurologic manifestations in every case conformed to the type designated by Redlich in 1917 as "encephalitis pontis et cerebelli," by Etter as "myelitis bulbi" or by Leyden as "acute ataxia."

Smears from the tonsils examined bacteriologically yielded no pathognomonic findings. Culturally, all cases showed the presence of pneumococci which were associated in two cases with Streptococcus haemolyticus and Staphylococcus aureus and in another case with a nonhemolyzing streptococcus. Cultures of the blood showed in one case, three weeks after the onset of the neurologic signs, S. aureus and a nonhemolyzing streptococcus; in another case S. aureus was found in the blood six weeks after the onset of the tonsillitis. Bacteriologic examination somewhat later showed that the blood was sterile in every case. Unfortunately the spinal fluid could be examined bacteriologically only in one case, and here the results were not conclusive.

In discussing the etiologic relationship between the encephalitis and the angina the author adds nothing that has not been published previously. In the absence of data taken at autopsy and of thorough bacteriologic investigations of the spinal fluid she offers two hypotheses as to the etiology: (1) that there may be a causal relationship between the present increasing number of cases of encephalitis and the epidemic of tonsillitis, and (2) that there exists an epidemic of encephalitis due to an unknown virus which seems to have a special affinity for the brain stem and needs another factor to activate it, and that another infection such as an angina may be the activating factor in a person who had previously been resistant to the virus.

The paper is concluded with an addendum, which contains the clinical reports of two more cases of the same condition observed by Schlesinger in the third medical division of the Wiener allgemeines Krankenhaus during July and October, 1931. Both patients recovered.

KESCHNER, New York. [ARCH. NEUROL. & PSYCHIAT.]

ROENTGEN IRRADIATION OF ADENOID VEGETATION. ZDENEK BENES, Časop. lék. česk. **69**:1495 (Oct. 31) 1930.

The author administered irradiation to a series of 138 patients over a period of two years. The only untoward results were a slight infiltration of the regional glands and slight pharyngeal hyperemia or a mild angina lasting only a few days. Both acute and chronic adenoid vegetations were treated. In all, 1,660 irradiations were given, and the immediate result was an improvement in respiration;

14 patients did not return for a check-up after the second irradiation, and these are not cited in the following figures. In 73 cases (mostly in children from 3 to 8 years of age) a complete cure was obtained; in 32 (children of from 10 to 14 years) improvement was obtained and in 19 it was not. In those in whom improvement occurred the entire reaction improved, even the facial expression. The treatment is especially recommended for recent or acute adenoid vegetations.

STULIK, Chicago. [AM. J. DIS. CHILD.]

FOCAL INFECTION OF TONSILLAR AND DENTAL ORIGIN. H. BJÖRN-HANSEN, Norsk mag. f. lægevidensk. **93**:378 (April) 1932.

The authors consider a leukocyte count and a sedimentation test to be of help in the diagnosis of focal infection. These tests may give negative results in spite of the existence of an infection, but the results are positive in the majority of cases.

Study of the patients whose cases are reported in this article has shown that focal treatment, that is, removal of tonsillar and dental foci, seems to prevent in many cases the development of secondary polyarthritis.

Focal treatment in the acute stage is not advisable, as aggravation of the disease may result.

Patients with focal infection are often hypersensitive to autogenic vaccine.

TOVERUD, Oslo, Norway. [AM. J. DIS. CHILD.]

Larynx

VENTRICLE OF LARYNX AND LARYNGITIS. K. GRAHE, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**:274 (Feb. 25) 1932.

Grahe recommends direct application of 2 per cent acriflavine hydrochloride to the ventricle of the larynx and the appendix ventriculi in subacute and chronic cases of laryngitis. Because of the anatomic construction of the appendix, which favors the accumulation of detritus, and the concentration of adenoid tissue in the ventricle and especially the appendix, this pocket is often the seat of infection and plays an important rôle in laryngeal inflammation. In laryngoscopy, a reddening and loosening of the mucosa adjoining the vocal cord and sometimes a covering of mucus in this region can be seen. In severe inflammation, the mucosa may occasionally be seen as a prolapse between the vocal and ventricular fold. The application of acriflavine hydrochloride is preceded by laryngeal anesthesia and is accomplished by means of special curved probes introduced into the ventricle. Symptoms of irritation may last two days afterward. One or two inhalations per day, without menthol or other irritants, are recommended for after-treatment. If necessary, the application may be repeated eight days after the symptoms of irritation have disappeared. This treatment results in marked improvement in cases of subacute or chronic laryngitis resistant to other therapy. If there is constant reinfection by secretions from the nose, mouth or bronchi, these sources of infection must, of course, be combated also. EDITOR'S ABSTRACT.

DIFFERENTIAL DIAGNOSIS OF EARLY STAGE OF SUPPURATIVE PERICHONDRITIS AND SUBMUCOUS LARYNGITIS. W. KLESTADT, Monatschr. f. Ohrenh. **66**:641 (June) 1932.

Klestadt discusses the diagnosis and treatment of the early stage of suppurative laryngeal perichondritis in connection with a case of severe phlegmonous suppuration of the larynx with mediastinal gravitation abscess in which the condition was mistakenly diagnosed and treated as fresh suppurative perichondritis laryngis. The diagnosis was based on a clinical picture of laryngeal stenosis, almost complete fixation of the vocal cords, ulcers in the intralaryngeal space, symptoms of an exterior thyroid abscess, absence of a primary pharyngeal abscess and an anamnesis revealing a ten day attack of influenza. Preliminary to the performance of cricotomy a large abscess in the submuscular layer over the cartilage was incised.

As only one small rough patch could be seen on the cartilage, the perichondritis was considered to be in an early stage of development. Appearance of pus on the inside of the thyroid cartilage a few days later and shedding of a small piece of cartilage with renewed abundant suppuration and general decline led to a second intervention. The perichondrium of the laminae of the thyroid cartilage was loose and easily stripped off; the surface of the cartilage was mottled. The lamellae of the cartilage were resected up to the point of normal adherence of the perichondrium. A gravitation abscess extending toward the back and down into the posterior portion of the mediastinum to the seventh rib was discovered. Following resection of the cartilage, the laryngeal symptoms disappeared rapidly. The gravitation abscess was healed by drainage with a catheter according to Heber, with occasional use of mechanical suction. Histologic examination failed to confirm the diagnosis of perichondritis, as the resected lamellae showed neither suppuration nor necrosis. The author thinks the red spots must have been hyperemic patches of the marrow, the yellow spots the surface of the bone exposed by edema and the white spots the surface of the cartilage. This case substantiates the opinion expressed by others that early suppurative perichondritis and submucous laryngitis cannot be differentiated. Therefore, intervention should at first be restricted to relief of stenosis. The cartilage or bone should not be attacked unless there is proof of a suppurative or granulated process within the perichondrium. It is a matter of question whether, until such proof is found, the treatment of suppurative perichondritis may not be the same as that of suppuration of the soft parts of the larynx.

EDITOR'S ABSTRACT.

Nose

PARANASAL SINUSES IN SCARLET FEVER. A. C. SILVERMAN, J. Pediat. **1**:38 (July) 1932.

Roentgen examination of the sinuses was made in 292 patients with scarlet fever. In the series 91 per cent showed shadows designated as "hazy to opaque," and in only 9 per cent were the sinuses reported as clear. All of 33 patients with otitis showed abnormal sinus shadows, while no patients with clear sinuses had any aural complications. The clinical diagnosis of sinusitis in children is not easy except in those that show swelling of the lids; hence the addition of roentgen evidence should be of importance.

Without desiring to draw unwarranted conclusions in the absence of control studies, the author raises the question whether sinus involvement in scarlet fever is not the rule rather than a complication.

LEMASTER, Evanston, Ill. [AM. J. DIS. CHILD.]

VASOMOTOR RHINITIS FROM THE ENDOCRINE STANDOINT. L. A. NELSON, Texas State J. Med. **27**:398 (Sept.) 1931.

Nelson presents a cursory review of the literature bearing on this subject and the results of treatment in twenty cases of his own. In selecting his cases he eliminated the cases of vasomotor rhinitis and hay fever in which the patients responded to therapy with calcium or with mineral acid. No attempt was made to separate the allergic from the nonallergic patients. Three groups were noted: (1) patients who showed a lowered metabolic rate and who were given thyroid, (2) patients with normal metabolic rates who showed a tendency to obesity, especially of the girdle type, and who were given pituitary (whole gland) and (3) the remainder of the patients, who were given parathyroid and calcium. Of the twenty patients fourteen had lowered metabolic rates and were relieved within from thirty-six hours to three days by thyroid; three patients with normal metabolic rates responded to pituitary (whole gland) therapy; three patients responded within twenty-four hours to the parathyroid-calcium therapy. In addition to the foregoing, ten patients with cedar fever, as determined by cutaneous

tests, who did not have their metabolic rates determined were given the first two remedies without relief but responded to a mixture of thyroid, parathyroid and calcium, usually within forty-eight hours. Case reports illustrative of each of the groups are presented. LUECKE, Dallas, Texas. [AM. J. DIS. CHILD.]

A CASE OF MUCOCELE OF THE MAXILLARY SINUS AFFECTING THE OCULAR GLOBE. Y. SHOJI, Ann. d'ocul. **169**:267 (April) 1932.

M. S., a man, aged 45, who acquired syphilis at 17 and, three years before the present observations, submitted to an operation for nasal empyema, noticed a certain amount of irritation about the left lower lid four months previous to examination. This lasted about three or four days, but since that time the region of the lower lid and cheek had progressively enlarged. He complained of binocular diplopia. Examination showed a swelling about the inferior part of the left lower lid. The tumor, of elastic consistence on the nasal side and of bony consistence on the temporal, occupied nearly the whole length of the lid and measured 18 mm. from above down. The left globe was displaced above and to the left, and the ocular movements downward were limited.

Under procaine hydrochloride anesthesia an incision was made over this mass, and a fibrous growth was found on the anterior surface of the maxillary sinus. On perforation of this area, a brown liquid escaped. After ablation of the tumor, one could see the whole cavity of the maxillary sinus. Convalescence followed in one week's time.

Cases of mucocele of the maxillary sinus have rarely been observed in ophthalmology. The mucocele enlarges the cavity and may cause inflammation of the retina or inflammation and atrophy of the optic nerve. The deviation of the ocular globe is the chief ocular symptom, which disappears on the opening and evacuation of the cavity. S. H. MCKEE. [ARCH. OPHTH.]

ETIOLOGY AND THERAPY OF VASOMOTOR RHINITIS. K. M. MENZEL, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**:345 (Feb. 25) 1932.

In a preliminary report on the study of basal metabolism in patients with the syndrome of vasomotor rhinitis, Menzel states that this syndrome, in a certain number of cases at least, may be regarded as an expression of a hyperthyroid overexcitability of the sympathetic nervous system. Roentgen irradiation of the thyroid seems to have a favorable influence on the disappearance of this syndrome in cases of the type mentioned. EDITOR'S ABSTRACT.

PHYSIOLOGY OF NASAL AND ORAL RESPIRATION: RÔLE OF NASAL SINUSES. W. DÖDERLEIN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:459 (May 13) 1932.

In investigating the physiology of nasal and oral respiration in human beings Döderlein found that the nasal conchas have no part in regulating the temperature of the inspired air, but that they regulate its moisture content; they retain a part of the water vapor of the expired air and thereby prevent desiccation of the nasal mucous membrane. The oral mucous membrane plays no rôle in the warming of inspired air; even in oral breathing the temperature regulation comes from the nose. Even when the nose is closed the air contained in the nose and the nasopharyngeal space takes part in the respiration. The air in the nasal sinuses was found to be warmer than the air in the nasal cavity. A series of experiments measuring the air pressure relations in the nasal sinuses led to the conclusion that this air takes part in the movement of the respiration air according to definite laws. With each inspiration a smaller or a larger amount of air, depending on the depth of breathing, passes from the nasal sinuses into the nose. At the end of inspiration the air from the nose again streams into the sinuses. In expiration more air from the nose is forced into the nasal sinuses so that at the end of

expiration the air pressure in the sinus is higher than that in the nasal cavity. In the pause before the next inspiration the air pressure in the nose and the sinus is equalized again. The author concludes that the physiologic significance of the nasal sinuses is that they constitute air reservoirs from which warm air, rich in oxygen, is constantly admixed with the inspired air according to need. Unused expiration air is stored in them.

EDITOR'S ABSTRACT.

NEW PLASTIC NASAL SURGERY. T. v. LIEBERMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:560 (May 13) 1932.

Liebermann describes a plastic operation on the nose undertaken to correct obstruction of nasal breathing. The nose of a man, aged 25, appeared too large, the base almost reaching the upper lip so that nasal breathing was possible only when the end of the nose was raised with the fingers. This condition was not due to the actual size of the nose but to the fact that the septum, which was too large for the nose, was constructed of such atrophic cartilage that it would not keep the nose in a normal position. The other cartilages of the nose were likewise so weak and soft that the nose sank downward and inward at its base. The septum was consequently crumpled and filled the nostrils, while the unsupported alae and tip of the nose closed over the nostrils. The operation undertaken by the author consisted in fastening the tip of the nose in such a way that it was in line with the end of the bony portion of the nose. A horseshoe-shaped incision was made beneath the glabella. The skin and the underlying tissue were tunneled as far as the tip of the nose, and an anchor shaped of gold wire was fitted into place and secured by a wire suture and overlayed with periosteum. Primary healing occurred. Nasal breathing has been unobstructed following the operation; the external scar is scarcely visible and the nose retains its lateral mobility.

EDITOR'S ABSTRACT.

Miscellaneous

OTOGENIC OSTEOMYELITIS OF PETROUS BONE, PHLEBITIS OF CAVERNOUS SINUS, CAROTID HEMORRHAGE. A. DEI ROSSI, Arch. ital. di otol. **43**:352 (June) 1932.

Dei Rossi cites a case of osteomyelitis of the petrous portion (petrositis) complicated with phlebitis of the cavernous sinus, carotid hemorrhage and terminal meningitis in the introduction to a review of the literature of carotid otorrhagia and of the pathogenesis, symptomatology and course of petrositis. The author distinguishes between two types of petrositis: (1) that found in the pneumatized petrous portions (deep osteitis), and (2) that in the spongy petrous portions (osteomyelitis).

EDITOR'S ABSTRACT.

CEREBRAL MANIFESTATIONS OF CIRCULATORY ORIGIN IN CONNECTION WITH EXCLUSION OF SINUS AND JUGULAR VEIN. P. OHNACKER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:1 (April 25) 1932.

Ohnacker reports several cases of sinus thrombosis in which cerebral symptoms developed as a result of exclusion of the route of venous discharge. The cases demonstrate the important part played in the development of these possible complications of sinus thrombosis by partial or total transfer of the venous discharge of the diseased side, whether by thrombosis or by surgical intervention, with concurrent constriction or transfer of the discharge channels of the healthy side owing to anatomic or pathologic causes, and, further, the part played by the transfer or exclusion of the transverse sinus by the possible influence of veins emptying into it. The cases also illustrate the confusing clinical pictures that may be presented by these complications. Sometimes elevation of the brain pressure is the foremost symptom; sometimes a beginning meningitis is simulated,

and, again, the symptoms may indicate a brain abscess. Restraint is to be exercised in regard to exploratory intervention in the brain in cases of sinus thrombosis complicated by cerebral manifestations, especially when the syndrome is not clear or does not correspond to any of the pictures of the other otogenous complications coming into question.

EDITOR'S ABSTRACT.

TREATMENT OF TUBERCULOSIS OF MUCOUS MEMBRANE BY FREEZING. R. NUSSBAUM and B. TEBRÜGGE, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:227 (May 23) 1932.

Nussbaum and Tebrügge think that freezing is the method of choice in the treatment of tuberculosis of the mucous membrane. They base their opinion on five years of experience with this form of therapy. Carbon dioxide snow mixed with acetone was used for the freezing, and the essential point of technic is the application of a sufficient quantity under sufficiently strong pressure for a sufficiently long time. The freezing of the mucous membrane heals the local ulcerative process in the shortest time with a certainty not approached by any other method even when the pulmonary tuberculosis is advanced and the general condition is very poor, as is usually the case in tuberculosis of the pharynx and the mouth. Scar formation takes place without destruction of tissue, leaving a smooth, soft scar and resulting in a minimum of functional disturbance. The painlessness of the freezing process, which is preceded by cocainization, is another advantage of this therapy. The authors experienced no recurrences among their patients. No complications or harmful general reactions were observed. The biologic effect of the freezing is still obscure.

EDITOR'S ABSTRACT.

DIAGNOSIS AND TREATMENT OF FOREIGN BODIES IN THE LOWER AIR PASSAGES AND IN THE ESOPHAGUS OF CHILDREN. K. AMERSBACH, Med. Klin. **28**:214 (Feb. 12) 1932.

Stenosis of the air passages or of the esophagus, if it appears suddenly, is always suggestive of foreign body. All possible diagnostic methods have to be used in this direction. An obscure form of bronchitis, particularly with foul-smelling sputum, should always suggest the possibility of foreign body. If all the other diagnostic methods fail, direct endoscopy has to be used. This is particularly true of esophagoscopy. Bronchoscopy is a somewhat more serious procedure. For diagnostic purposes bronchoscopy only of the upper passages is permissible. For removal of foreign bodies, bronchoscopy of the lower passages is often unavoidable, chiefly because the long-continued tracheoscopy usually leads to edema of the glottis which finally makes tracheotomy necessary. All endoscopic instruments have to be in perfect shape, as otherwise in place of one foreign body two may result. The old type of instruments, like the coin forceps, are entirely out of place in this era of esophagoscopy. Rectal anesthesia with avertin is the method of choice for endoscopic procedures.

ZENTAY, St. Louis. [AM. J. DIS. CHILD.]

Directory of Otolaryngologic Societies *

INTERNATIONAL

Second International Otolaryngological Congress

President: Prof. Dr. A. G. Tapia, Hermosilla, 2, Mod, Madrid, Spain.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde, Heereweg 143, Gronigen, Holland.

FOREIGN

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

Royal Society of Medicine, Section of Laryngology

President: Dr. Walter G. Howarth, 21 Devonshire Place, W., London.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.

Royal Society of Medicine, Section of Otology

President: Dr. Norman Patterson, 82 Portland Pl., W. 1, London.
Secretary: Dr. T. B. Jobson, Netherwood, Epsom Road, Guildford.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Milwaukee. Time: June 12-16, 1933.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.

American Bronchoscopic Society

President: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Edwin McGinnis, 104 S. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. George Fetterolf, 2010 Spruce St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Washington, D. C. Time: May 9-11, 1933.

American Laryngological, Rhinological and Otological Society

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.
Place: Chicago. Time: June 8-10, 1933.

American Otological Society

President: Dr. George L. Tobey, Jr., 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.
Place: Washington, D. C. Time: May 9-11, 1933.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. Seventh St., Los Angeles.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

Southwestern Michigan Triological Society

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Don C. Lyon, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. Harry L. Whitaker, 1612 Fremont St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. M. H. Gill, 36 Pearl St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. W. J. Marshall, 212 N. Higgins Ave., Missoula.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Time: Semi-annually.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. McWhorter, 106 E. Earle St., Anderson.
Secretary-Treasurer: Dr. G. M. Truluck, Orangeburg.
Time: April and December.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.
Place: Fort Worth. Time: Dec. 2-3, 1932.

Utah Ophthalmological Society

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

Virginia Society of Otolaryngology and Ophthalmology

President: Dr. Frank P. Smart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. Henry Grant Preston, Professional Bldg., Harrisonburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore City Medical Society, Section on Otology and Laryngology

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. John F. Healey, 925 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Herrmann E. Bozer, 468 Delaware Ave., Buffalo.
Place: Buffalo Club. Time: Second Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Charles Younger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

COLUMBUS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Ia.
Time: Third Monday of each month, September to May, inclusive.

DETROIT OTO-LARYNGOLOGICAL SOCIETY

President: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. J. M. Sutherland, 8-257 General Motors Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6.30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 314 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month, September to May, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Edward Leo Berger, 903 Park Pl., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OTOLARYNGOLOGY

Chairman: Dr. Charles Imperatori, 108 E. 38th St., New York.
Secretary: Dr. David H. Jones, 140 E. 54th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., third Wednesday of each month from October to May.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

PHILADELPHIA LARYNGOLOGICAL SOCIETY

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

PITTSBURGH OTOLOGICAL SOCIETY

Chairman: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.
Secretary-Treasurer: Dr. Alexander R. Hampsey, 806 May Bldg., Pittsburgh.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS EAR, NOSE AND THROAT CLUB

Acting President: Senior member of the executive committee: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Charles J. Boehs, 1126 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.